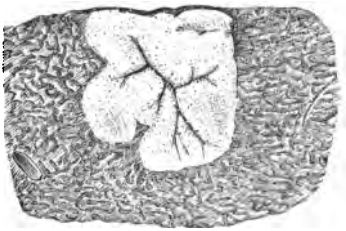

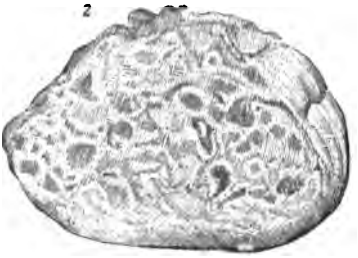

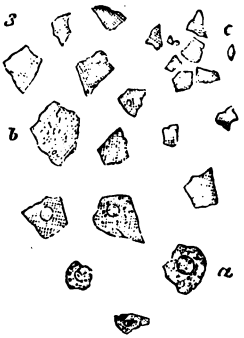

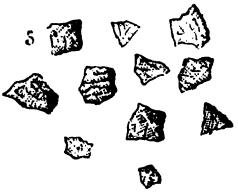

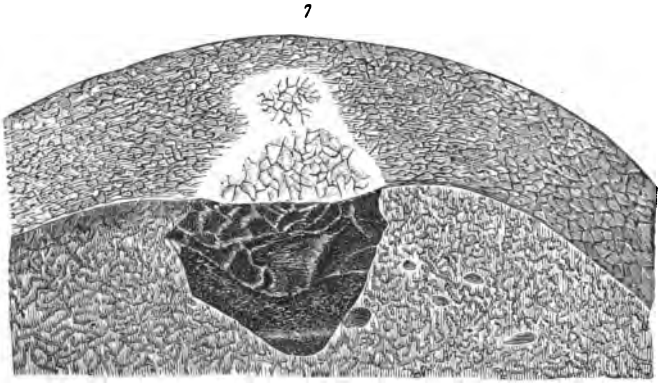

FRERICHS ON DISEASES OF THE LIVER.

A

CLINICAL TREATISE

ON

DISEASES OF THE LIVER.

BY

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IN THREE VOLUMES.

VOL. II.

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EXPLANATION OF FRONTISPIECE.

Fig. 1. Syphilitic fibroid nodule of liver. (See p. 161.)—Circumscribed yellowish-white infiltration, from the liver of a syphilitic patient. The abnormal deposit is situated at the periphery of the organ, where it is sharply defined from the surrounding hyperæmic tissue. It is of firm consistence and consists of connective tissue infiltrated with a crumbling amorphous material, nuclei and oil-globules. In the centre of the nodule are the ramifications of the penetrating blood-vessels, and close to them are some soft yellowish-green places.

Fig. 2. Alveolar cancer of the liver. (See Vol. III., p. 51.)—*a*. Serous investment of outer surface. *b*. Under surface. *c*. The morbid growth growing from the upper surface of the liver.

Figs. 3, 4, 5, and 6. Hepatic cells in a state of waxy or amyloid degeneration. (See p. 173.) Fig. 3. A group of hepatic cells, in which the different stages of the deposit of amyloid matter may be traced.—*a*. A cell with a distended nucleus; *b*. An enlarged cell, uniformly filled; the nucleus is no longer visible; *c*. Crushed débris of cells. Fig. 4. Change of color in the cells produced by the addition of solution of iodine. Fig. 5. The same after the addition of solution of iodine and sulphuric acid. Fig. 6. A firm aggregation of hepatic cells colored by solution of iodine.

Fig. 7. Cavernous tumor of the liver. (See Vol. III., p. 1.)—Portion of the right lobe of the liver, showing a cavernous tumor (Telangiectasis) with sharply defined margins, penetrating in a wedge-shaped form, from the outer surface into the parenchyma, of the natural size.

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A CLINICAL TREATISE

ON

DISEASES OF THE LIVER.

CHAPTER I.

THE PIGMENT-LIVER. — MELANÆMIC LIVER. — ALTERATIONS IN THE LIVER RESULTING FROM INTERMITTENT FEVERS.

1. *Historical Account.*

THERE is an old tradition, that black substances are formed in the spleen and in the blood of the portal vein, and may give rise to disease; the so-called black bile constituted an important element of the earliest theories of humoral pathology. Galen thought that this substance accumulated in the spleen, as a product of the formation of bile, and that from that locality it gave rise to obstructions of the vessels, enlargements (*Anschoppungen*) of the abdominal viscera, and dangerous nervous symptoms. This theory, with a few intermissions (*Van Helmont*, *Sylvius*), and with some slight modifications, continued to be resorted to for the explanation of abdominal affections generally, and of diseases of the portal system in particular. The mode of origin, and the pathological importance of atrabiliary matter, we find discussed in detail by Boerhaave[1] and Van Swieten.[2] These observers were of opinion, that owing to morbid influences of a varied nature, the fluid elements of the blood were diminished, and the solid constituents condensed into a black, fatty, earthy substance.[3] This substance, it was believed, in virtue of its peculiar nature, accumulated in the portal vein, stagnated here for a long period, assumed acrid and corrosive characters, deranged the functions of the abdominal viscera, and ultimately was converted into black bile.[4] This black bile was then set in motion by certain exciting causes, and, penetrating into the liver, the heart, the lungs, and the brain, produced most serious derangements everywhere:—at one time fever, when the substance had become putrid;

[1] Aphorismi de cognosc. et curand. morb.
[2] Comment. ad Boerhaav. aphorism. Tom. III., p. 461, *et seq.*
[3] § 1092.
[4] § 1098, 1102.

at another time, when it led to mechanical obstruction of the cerebral vessels, dangerous symptoms of another character, such as convulsions, paralysis, delirium, &c.[1]

The theories in question received a considerable expansion from Kämpf,[2] who added to the atra-biliary matters a series of other substances, which he regarded as proceeding from the blood-plasma.

It was not until the end of last century that Riel, who in this, as in so many other questions, anticipated the age in which he lived, showed how the doctrine of black bile was opposed to physiological observations.[3] After him, Heusinger[4] attributed the morbid condition under consideration to a deposit of black pigment, and, along with Puchelt, accounted for it by an exaggerated venous condition of the blood. The majority of physicians, however, continued, as before, to believe in the dreaded pathogenetic potency of the black bile. The marsh fevers of the tropics, and the diseases which in 1826 devastated the littoral districts of the northwest of Germany and the Netherlands, were regarded as atra-biliary fevers. Even in the year 1829 Vogel described the *atra bilis* after the fashion of the ancients, in the Berlin Encyclopædia.

Scientific medicine had scarcely removed this traditional remnant of a Hippocratic humoral pathology, when it became necessary, from an unprejudiced observation of facts, to relapse into the old doctrine. Diseases became known in which black matter produced by decomposition of the blood, became developed in the spleen, which black matter passed into the portal vein, and at one time obstructed the hepatic vessels, at another, passed through these, and, entering the general circulation, filled up the capillaries of the brain and other organs,—morbid processes, in fact, which gave rise to symptoms similar to those which were described by the ancients.

It is only in the most recent times that accurate observations have been made of this lesion, although isolated instances are found recorded by the older physicians. Long ago, Lancisi[5] found the liver of an individual who had died of bilious fever, of a blackish tinge; and Stoll[6] described a dark pigmentary deposit in the brain and liver of a female who had succumbed after several attacks of fever. The statements made by Bailly[7] in his "Pathological Anatomy of Intermittent Fever," are more copious and to the point:—"Le foie tout entier était noirâtre, semblait composé de sang noir," &c.—"la couleur du cerveau beaucoup plus foncée." Simultaneously, Billard described the same change in the brain of three patients who died of acute cerebral disease;[8] and observations of a similar nature were found in Montfauçon's "*Histoire médicale des marais*" (pp. 306-322).

Black pigmentary deposits were repeatedly observed in the spleen, liver, and brain, during the fever epidemic which, in 1826, raged along the coast of the North Sea.[9]

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[1] § 1104.
[2] *Op. ant. cit.*, p. 14.
[3] Riel. Memorab. clinic. med. pract., Fasc. III., p. 54.
[4] Untersuchungen über anomale Kohlen- und Pigmentbildung im menschlichen Körper. Eisenach, 1823.
[5] De noxiis paludum effluviis.
[6] Ratio medendi, Tom. I., p. 196.
[7] Traité anat. patholog. des fièvres intermittentes. Paris, 1825, p. 181, &c.
[8] Archiv. gener., 1825.
[9] POPKEN, Historia epidem. malignæ Jeveræ observ. Bremæ, 1827. FRICKE, Bericht über seine Reise nach Holland im Jahre 1826.

Richard Bright, in his "*Reports of Medical Cases*," published in London in 1831 (Plates XVII., XIX., and Chap. CI.), gives a masterly representation of a brain, the cortical substance of which was of a dark color, like black-lead.[1] This was taken from a man who died of cerebral paralysis, after an attack of fever. Physicians, who have had an opportunity of observing intermittent and remittent fevers in hot climates, have frequently described a black tinging of the spleen and liver; particularly Annesley,[2] Haspel,[3] Stewardson,[4] and others.

All these observations remained without further results, because the mode of origin and distribution of the pigment were not accurately traced. Not until the year 1837 did Meckel[5] ascertain that the dark color of the organs depended upon an accumulation of pigment in the blood; two years later, Virchow found numerous pigment cells in the blood and in the enlarged spleen of a man who became dropsical after a persistent intermittent fever.[6] Heschl[7] and Planer[8] have recorded numerous observations of a similar nature.

II. *Anatomical Description of Pigment-Liver, and of the co-existing conditions of the Spleen, Brain, Kidneys, &c.*

In individuals who die from the effects of marsh poison, under symptoms of severe intermittent, remittent, or continued fevers, we frequently find peculiar changes of the liver associated with functional derangements of the organ, and of the parts pertaining to the portal system. The liver presents a steel-gray, or blackish, or not unfrequently a chocolate color; brown insulated figures are observed upon a dark ground. This change of color is produced by pigment-matter which is accumulated in the vascular apparatus of the gland. In fine sections of the hardened tissue, accumulations of pigment may be observed in the capillary network of the portal and hepatic veins, as also in the larger branches of these vessels: these deposits are either uniformly distributed, or limited, for the most part, to certain regions. Sometimes the brownish-colored lobules appear surrounded by black margins, owing to the interlobular vein being filled with colored particles (Fig. 1); in general, however, the pigment-matter is more uniformly distributed, extending from the circumference of the lobules halfway to their centre, or penetrating as far as the commencements of the hepatic veins, or, still farther, into the vena cava.

In most cases, the arterial system is implicated as well as the venous, the branches of the hepatic artery containing large quantities of black coloring matter.[9] The hepatic cells remain exempt; in no case have I observed any deposit of pigment in them, such as is described by Virchow.[10]

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[1] "It was almost of the color of black-lead."

[2] *Op. cit.*, vol. II., p. 482:—"Liver of very dark color."

[3] Maladies de l'Algérie, I., 335; II., 318.

[4] The American Journal, April, 1841, p. 42.

[5] Zeitschr. für Psychiatrie von Damerow, 1847; also, Deutsche Klinik, 1850.

[6] His "Archiv für Patholog. Anatomie," 1849 and 1853.

[7] Zeitschrift der Gesellschaft der Aerzte in Wien, 1850.

[8] The same for 1854.

[9] Not only in malarious fevers, but very frequently, also under other circumstances, as in cirrhosis, cancer, and echinococci of the liver, the hepatic artery contains black pigment, apparently owing to interruptions in the capillary circulation of this vessel.

[10] After extravasations of blood into the hepatic parenchyma, red, brown, and black deposits of pigment in the cells are not unfrequently observed; in one case of cirrhosis I met with extensive masses of this nature.

They are either normal in their characters, or filled with brown bile; they are often infiltrated with oil, and occasionally, but only after a long continuance of the disease, they contain colloid or lardaceous matter. In the acute cases, the size of the entire organ appears either normal or enlarged; the gland is swollen from congestion, and here and there, also, it is infiltrated and softened by extravasations of blood; at a later period, it diminishes in volume, and there frequently ensues a true atrophy, provided that the gland does not become infiltrated with colloid matter,—an occurrence which I have only met with in rare instances.

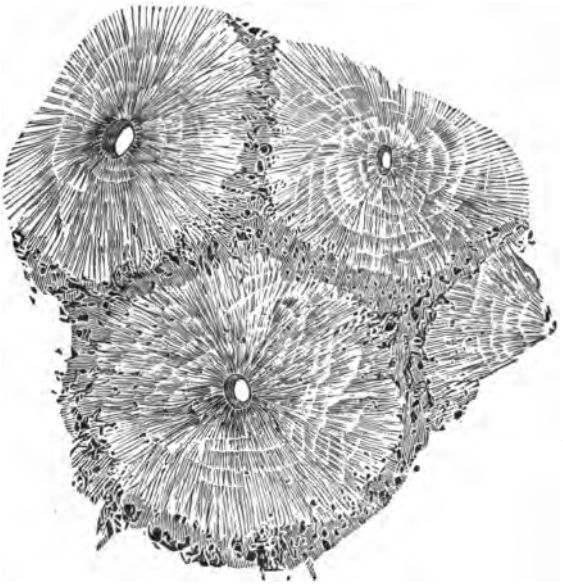

FIG. 1.—Fine section of a pigment-liver, previously boiled and treated with a solution of potash; magnified 90 diameters. The pigment is seen to be deposited, for the most part, in the interlobular veins, forming a black zone surrounding each lobule, and to have penetrated but very slightly into the interior of the lobules. In most cases, the pigment is distributed more uniformly than is here represented.

Along with these abnormal conditions of the liver, we constantly find similar appearances in the spleen. This organ is likewise dark-brown, or sometimes bluish-black; at one time it presents a uniform color, and at another, is speckled; in its parenchyma large quantities of the same pigment can be detected as is present in the liver. At the same time, the size and consistence of the organ become altered; in the acute cases of the disease, it is usually soft, congested, and considerably enlarged; in the less severe cases, there is usually only a slight alteration in the volume of the organ, unless, as seldom happens, it undergoes lardaceous degeneration; in this case, its volume and consistence are considerably increased. Analogous changes are observed in the lymphatic glands.

The liver and the spleen are the parts in which the black pigment is most constantly observed under the circumstances just mentioned. Very frequently, however, there are other organs implicated as well, the pigment, when it passes in large quantity into the circulation, being carried to them by the current of the blood.

A large quantity has usually been found in the capillaries of the lungs;

in old individuals, however, in whom pigmentary deposits of another nature take place in these organs, it is difficult to distinguish between the two.

This tinging with pigment is much more easily detected in the brain, in which considerable collections are sometimes recognized by the dark-coloring of the cortical substance. This assumes a chocolate or black-lead-like hue, whilst the white matter remains unchanged; it is only when the pigmentary deposit is excessive that we see the white matter present a gray appearance, and the fine vessels in it resembling brown streaks. Under such circumstances, microscopic examination has shown the capillaries to be filled with black granules and scales (*Schollen*), which at one time are uniformly distributed, but at another are aggregated in groups. Frequently we observe along with the pigment scales a pale hyaline coagulum, which when viewed with a light of a strongly refracting power, may be seen to fill up the calibre of individual capillaries.

The kidneys frequently participate in the pigmentary deposit to a remarkable extent. When this is the case, gray spots make their appearance in the cortical substance; and in a few cases, we may observe dark lines in the pyramids following the course of the blood-vessels and uriniferous tubes. Under the microscope we can detect the pigment-matter in the capillaries of the cortical substance, and particularly in the Malpighian bodies; now and then we find isolated little fragments in the uriniferous tubes.

The remaining organs and tissues, such as the external integument, the mucous membranes, the muscular tissue, &c., by no means remain exempt, as may at once be seen by a simple inspection of their gray tint; but the accumulation is seldom so considerable in these parts as in the organs previously mentioned, and it appears to be of subordinate importance, owing to the comparatively slight physiological value of these structures. In the typical forms of the disease we find, as a general rule, pigment deposited everywhere where the blood penetrates, and independently of the abdominal glandular organs, we find it in greatest abundance in those parts of the body where the capillaries are most narrow, and consequently where the pigment flakes are most easily arrested.

The pigment which is found in the organs and tissues, is brought to them by the blood;[1] it exists in abundance in this fluid, and particularly in that portion of it contained in the portal vein, where we can most easily study its properties, color, and chemical relations. The usual form in which the pigment makes its appearance is that of small, rounded, or angular granules, which at one time are sharply defined, and at another are surrounded by a brownish or pale margin. These granules are occasionally isolated, but most frequently several of them are connected together in groups by a pale substance, soluble in acetic acid and in caustic alkalies. In form they are rounded or elongated, sausage-shaped or irregularly branched. They have no defined membranous outline; the hyaline connecting-substance which presents the characters of fibrinous matter, forms sometimes a broad, and sometimes a narrow rim, without any sharply-defined outline.

True pigment cells are observed along with the granules and granular masses, though in somewhat smaller quantity. These partly resemble in form and size the colorless corpuscles of the blood, and partly consist of

[1] In the capillaries of the hepatic artery, and occasionally also in those of the portal vein, the pigment appears to lie in the walls of the vessel.

larger spindle-, or club-shaped cells with rounded nuclei, and sharply-defined walls, like the cells which we are wont to find, along with free granules, in the spleen. These cells contain a greater or smaller number of black granules. Planer was unable to convince himself of the presence of these pigment cells, which Virchow had previously described; I have seldom failed to find them in the blood of the portal vein; in most cases it is easy to avoid confounding these cells with the granular masses, which are bound together by fibrinous material. (Fig. 2.)

Besides the forms just described, larger fragments of pigment are observed, which, for the most part, have an irregular form, and upon pressure, look as if they had been broken off from still larger masses. Sometimes these fragments appear to be cylindrical, and are bounded laterally by two straight parallel lines, whilst their extremities are irregularly broken off; so that they remind one of the appearance of the smaller vessels, of which they appear to be casts. Their size is not unfrequently considerable; I have seen some which were $\frac{1}{100}$ of a line in breadth, and $\frac{1}{20}$ of a line in length. They are usually surrounded by a rim of pale transparent substance of greater or less breadth; sometimes this rim is only visible on one side. (Fig. 2.)

The color of the pigment is, for the most part, deep black, more rarely it is brown or ochre-colored, and least frequently of all, reddish-yellow. These different colors represent the various stages in the transformation of the red pigment of the blood into melanotic matter. The gradually advancing metamorphosis, however, manifests itself not merely in the color, but also in its relations to re-agents. The resistance offered by the different sorts of pigment to the action of acids and caustic alkalies is very variable. The more recent products are rendered pale, and lose their color with greater or less rapidity; those, on the other hand, of an earlier date, resist the action of these agents for a long time; I have not unfrequently permitted the latter to be acted on for days by caustic soda, without their color being destroyed.

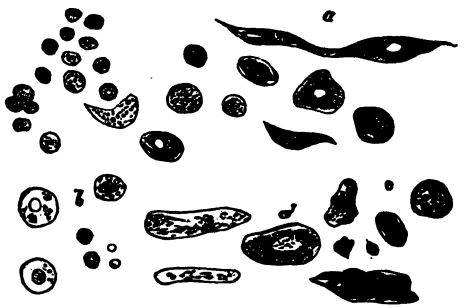

FIG. 2.—Pigment-matter from the portal veins.
a, From the trunk of the vessel. The epithelium of the lining membrane containing black pigment, and some of the cells with a distinct rounded or spindle-shaped nucleus; the younger cells were colored reddish-brown; normal blood corpuscles.
b, Cylindrical scales (*Schollen*) containing pigment.
c, Fragments of black pigment, enveloped in a hyaline substance, from the splenic vein.
a', From the splenic vein.

Along with this pigment, coagula of a hyaline character, and free from coloring-matter, are found in the blood. These resemble, in their form, the last-described variety of pigment flakes; from the fact of their being colorless, they are easily overlooked. Nothing unusual can be detected in the blood-corpuscles; sometimes the number of the white corpuscles appears to be increased,—a fact long ago observed by Meckel, but by no means of universal occurrence; in the majority of cases, and especially in those of an acute nature, nothing abnormal can be made out with regard to the corpuscles.

Such are the *post-mortem* appearances found in melanæmic individuals. The first question which now forces itself upon us, is:—Whence, and in

what way does this pigment originate?—and the second:—What are its results upon the functions and textures of the individual organs?

III. *Place and Mode of Origin of Pigment.*

This question has been answered by most observers, to the effect that the spleen constitutes the seat of formation of the melanotic matter. There are many grounds for such an opinion, but no proofs exist which can claim this function for the spleen exclusively. Experience shows that the transformation of the red matter of the blood into black pigment may take place everywhere throughout the vascular system, and also external to it. Although the spleen, from the nature of its structure and circulation appears pre-eminently adapted for such a purpose, we must keep in view, *à priori*, the possibility of the same blood metamorphoses taking place in other parts of the body, and we cannot arrive at the conclusion that these changes are exclusively confined to the spleen, until observation lends no support to the participation of other parts of the vascular system.

There can be no doubt, however, that the largest portion of the pigment is formed in the spleen, that it passes from this organ into the portal vein, and that part of it remains impacted in the capillaries of the liver, whilst the rest passes through these capillaries, and is carried into the general circulation.

There are many facts which may be appealed to in favor of such an hypothesis. It is known, that in the spleen of men and animals, and particularly in the spleen of the naked amphibia, cell-like structures are met with, even in the healthy state, which contain blood corpuscles or pigment molecules. It is true, their similar formations are occasionally found in other parts of the body, as in extravasations of blood in the brain, but nowhere so generally as in the spleen. In melanæmia, the accumulation of pigment is not so constant in any organ as in the spleen;—this is a rule almost without exception. Next to the spleen, as regards the frequency and intensity of the deposit of pigment, comes the liver, and after this the remaining organs, the lungs, the brain, the kidneys, &c. Cases are not unfrequent, where only the spleen contains a quantity of pigment, and others, in which the spleen and liver alone are infiltrated with dark coloring-matter, whilst the other organs preserve their normal hue; but I have never observed an accumulation of pigment in the blood of the heart, in the capillaries of the brain, kidneys, &c., without the large glandular organs of the abdomen being implicated. Another argument, in favor of the origin of the pigment of melanæmic patients in the spleen, is the form in which the black pigment is found in the blood. In portions of blood-clot, I have observed the pigment contained in the same elementary structures as are present in the splenic pulp, colorless blood corpuscles, with a simple or divided nucleus, and spindle- and club-shaped epithelium cells from the cavernous sinus of the spleen. The supposition that other organs are implicated in the formation of pigment rarely finds any support from examination. The epithelium of the endocardium, and of the lining membrane of the vessels in different regions of the body, does not present any unusual appearance; the accumulation of pigment in the liver, the brain, the kidneys, &c., is always limited to the finest capillaries; extensive deposits which might cause the blood to become overloaded with pigment

are nowhere to be found. Only in one case have I been able to ascertain with certainty, that the formation of the black pigment is not exclusively confined to the spleen, but occurs also in the liver. In an individual who died of exhaustion, after a protracted quartan fever, complicated with albuminuria, the enlarged and lardaceous spleen was completely free from pigment, whilst considerable quantities were accumulated in the liver. Not only did this pigment fill the finer capillaries of the portal and hepatic veins, but it also extended into the larger branches of the portal veins; these were blocked up at many places with black, friable coagula, and might be easily traced with a pocket lens, or with the naked eye. Besides the liver, the parenchyma of the kidneys contained much pigment, whilst that of the brain remained free.

Hence, it would seem that the usual source of the formation of pigment is the spleen, and that, along with it, but only in exceptional cases, other organs, and particularly the liver, participate.

It is a more difficult matter to reply to the other question, which has reference to the manner in which this formation of pigment takes place. We shall not solve this problem until we possess a more intimate knowledge of the minute structure of the splenic pulp than we do at present. If we assume, what in the present state of the question appears the more probable view, that the capillaries of the splenic artery pour their blood into a system of large sinuses, whence it passes into the efferent veins, the following explanation of the production of the pigment might be offered. Even in the normal state, the blood which has flowed rapidly through the narrow capillaries, would pass more slowly through the ample channel of the venous sinuses, and not unfrequently would stagnate in some places, so that conglomerate masses of blood corpuscles would be formed, which gradually might be transformed into pigment. In the intense congestions which experience shows to attack the spleen as a consequence of intermittent fever, these stagnations are very great, and would lead to considerable accumulation of pigment. According to this view, the pigment would arise from the stagnation of the blood in the venous sinuses of the spleen; the club- and spindle-shaped pigment cells are the epithelium of the lining membrane of the sinuses infiltrated with the decomposed red matter of the blood; the globular pigment cells are colorless blood corpuscles, infiltrated with molecules of coloring-matter, and the pigment scales (*Schollen*) are the broken-up fragments of the coagulum. Why the formation of pigment is either absent or less marked[1] in many other congestions of the spleen, in typhus, pyæmia, and simple intermittent fever, why it is that the metamorphosis of the blood-pigment into melanotic matter, apparently takes place much more rapidly here than at other places, are questions to which at present we cannot give a satisfactory reply. The acid character of the splenic pulp, however, must exercise an important influence over the metamorphosis of the blood-pigment.

Another point of importance for the clinical elucidation of the consequences of this morbid process, would be the tracing of the chemical products of metamorphosis which are formed by the destruction of the blood corpuscles, the morphological residue of which presents itself in the form of pigment. It is scarcely to be imagined, that amid so great a destruction of the elements of blood, products of metamorphosis should not be formed, concerning which the microscope can furnish no information. These are carried along with the pigment into the circulation, and will be

[1] See Observations No. XIII. and XVI., Vol. I., pp 121, 147.

taken into consideration in explaining the nervous symptoms, which we know to accompany malignant intermittent fever.[1]

IV. *The Results produced by the formation of Pigment upon the Structure and Functions of the various organs.*

The effects upon the system of the morbid process taking place in the splenic parenchyma which has just been described, are of a complicated nature, and can only be understood by a close investigation of the individual agents which are concerned in them.

The destruction of large numbers of the blood corpuscles contributes, in the first place, to the production of a state of the blood like that of chlorosis, which usually soon makes its appearance in the course of intermittent fevers. This effect upon the general mass of the blood is aggravated by the interruption of the important blood-forming function of the spleen which takes place at the same time. It is a difficult matter to determine to what extent each of these agencies is calculated to act; the loss of blood corpuscles, however, must be proportioned to their destruction, and consequently to the quantity of pigment which has been formed.[2] An increase of the colorless corpuscles, as a consequence of the disease of the spleen, is not observed: in most cases the examination of the blood has shown no remarkable deviation in this respect.

The pigment originating in the spleen, passes first with the blood of the portal vein into the liver. It is here that the first functional derangements are observed. One portion of the pigment passes through the capillaries unarrested, and enters the general circulation; the larger particles remain impacted in the capillaries of the portal vein, and obstruct the circulation of the blood through these vessels. The accumulation of pigment is sometimes seen to take place chiefly at the periphery of the lobules in the interlobular veins; but at other times it extends throughout the entire capillary vascular system, and penetrates into the interior of the lobules as far as the central veins. The derangements of the circulation and their consequences vary according to the quantity of the large pigment scales and cells.

These derangements manifest themselves first in an abnormal secretion of the liver. The bile is usually secreted in large quantity, and we have repeatedly found in it considerable quantities of albumen; leucine can al-

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[1] Boerhaave and Van Swieten (*loc. cit.*, III., p. 496) long ago assumed the existence of a putrefaction of the atra-biliary matter, and attributed the symptoms which were induced by this, partly to the pollution of the blood by the products of putrefaction, and partly to the mechanical obstruction of the capillaries by the black masses. It is obvious that the detection of these products was not to be thought of in the condition of chemical science at that time. Even at the present day, the same difficulty presents itself in explaining the problem before us, because the intermediate substances which arise from the decomposition of the albuminous principles have no characteristic properties, and cannot be detected until the decomposition has arrived at certain ultimate products. Such being the case, negative results cannot decide the question.

We have repeatedly examined the spleen of melanæmic patients, and have found numerous products of metamorphosis which are developed in large quantities in this organ, but have failed to detect any characteristic new substance foreign to the organ.

[2] The loss in this way may be very considerable. Occasionally the organ, which is almost black from the accumulation of blood, is considerably enlarged, diminished in its consistence, and, at some parts, infiltrated with extravasations of blood.

ways be detected in the hepatic parenchyma; its saccharine constituents, however, are unchanged.

Extensive capillary stagnation gives rise to obstruction of the circulation of the blood in the roots of the portal vein, which manifests itself in various ways according to its intensity. Sometimes we see exhausting hæmorrhages from the gastro-intestinal mucous membrane coming on in an intermittent manner, or more frequently, profuse diarrhœas, occasionally associated with vomiting, &c.; acute serous effusions into the peritoneal sac, together with bloody suffusions of the serous membranes, constitute another result; while at a later period, chronic atrophy of the liver, with its train of consequences, may make its appearance.

The next organ, in point of frequency to the liver, which undergoes important organic and functional derangements, is the brain. Numerous particles of pigment, which have passed unarrested through the vessels of the liver and of the lungs, accumulate in the narrow capillaries of this organ, and particularly in those of the cortical substance. Even by simple inspection of the shade of color, we can form an approximative notion of the quantity of coloring-matter which has been deposited, and of the extent of the vascular obstruction. We must not, however, rely entirely upon inspection, for slight accumulations of pigment in the capillaries easily escape notice, particularly when viewed by an unpractised eye, and can only be distinguished with the assistance of the microscope. In addition to the above, it is not at all uncommon for the vessels to become obstructed by a colorless fibrinous-like coagulum, which of course does not affect the shade of color. The mechanical interruption to the circulation which is produced in this way, not unfrequently gives rise to rupture of the small vessels, and the formation of numerous capillary apoplexies. Meckel long ago made observations of this nature; Planer described eight cases in which small extravasations were scattered through the gray and white substance of the brain. These numerous hæmorrhages have not come under my own observation; but in two cases I have observed extravasation into the meninges.

It has not been proved by direct examinations whether, besides hæmorrhages, other organic lesions of the brain, such as atrophy from interrupted supply of plasma, result from occlusion of the capillaries.

I have seen pigment brains of old date without any remarkable diminution of the cortical substance. Other functional derangements, indicative of material changes in the cortical substance of the brain, have only occurred to me in three cases; these cases, however, I only saw in a cursory manner during a journey in Poland.[1]

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[1] One of these was a lady in her fortieth year, who, after an attack of quotidian fever, accompanied by somnolence, suffered from protracted loss of memory. The functions of vegetative life resumed their normal condition, and there were no derangements of motion or sensation present. The headache and giddiness gradually diminished after the removal of the intermittent fever by means of quinine; but the weakness of memory and the inability to find suitable words for objects and ideas were still on the increase two months after the cessation of the ague.

Another case was that of a girl, aged 9, living in the same district, where, according to the evidence of the medical men, intermittent fever, terminating fatally, was at the time very prevalent. This girl, whose mental powers had previously been normal, had undergone several attacks of tertian fever. After a protracted use of preparations of bark, she recovered in her bodily symptoms; but her mental faculties gave way, and a state of complete idiocy, accompanied by a ravenous appetite, supervened.

In these two cases, it is uncertain whether atrophy of the brain had resulted from occlusion of the capillaries, or whether it had been induced by the extensive capillary

Along with the structural changes of the brain which have just been described, remarkable aberrations of the functions of the organ are usually observed during life.

These derangements vary in their nature; sometimes they intermit, and cease with each paroxysm of fever, but more frequently they are protracted into the period of the intermission, or are continuous. Even in the last case, however, there are usually unmistakable indications of more or less distinct remissions.

The nature of the cerebral disturbances manifests itself in very various ways; in the slighter cases, they assume the form of headache and giddiness; in the more severe, that of delirium, but more usually of coma, and not unfrequently, there are disturbances of mobility, convulsions, or paralysis. The most constant symptom is dull pain in the head, extending over the entire vertex, and almost always associated with attacks of giddiness. This pain was observed in every case in which the loss of consciousness had not arrested all power to communicate subjective sensations; occasionally, the pain was so severe as to cause the patients to utter loud cries. The headache was frequently accompanied by derangements of the senses, noises in the ears, deafness, black vision, and dimness of sight, and occasionally by nausea, a tendency to vomit and vomiting.

Delirium is less frequent; sometimes it is quiet, like that usually met with in typhus; but at other times it is violent, and associated with great excitement and restlessness, so that it is necessary to fasten the patient in bed. By degrees the excitement passes into somnolence and deep coma, which is the most usual form of cerebral disturbance. In several cases giddiness, without any headache, has existed for a long period after the removal of the fever, and this has even been of such intensity that the patients have fallen down suddenly while walking, while, at the same time, the anæmia has not been so great as to account for the occurrence. (*Febris vertiginosa Paccinotti.*)

The slight forms of somnolence from which the patient can be roused by loud calling, so as to give rational answers, in most cases after a short time lapse into deep stupor. This stupor sometimes ceases during the intermission, to return with the next paroxysm.

Abnormal conditions of mobility, convulsions and paralyses are much less frequent than derangements of the mental faculties. I have met with convulsions in eight cases; at one time they appeared as slight spasmodic movements of individual muscles of the trunk and extremities, at another time as rapid, rotating, or tossing movements of the extremities and head, and, again, as general convulsions, similar to those of epilepsy, which usually lasted from five to ten minutes, and returned after a shorter or longer interval. Paralysis was only present as an exception. It attacked at one time the muscles concerned in articulation or swallowing, and at another, the extremities; in the latter case, the paralysis was either double or confined to one side. In one case I have seen the paralysis make its appearance suddenly, and here capillary hæmorrhage was found;

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apoplexies consequent upon this occlusion, or whether the intermittent fever was complicated with other accidental changes in the brain.

It is worthy of notice, that Sydenham (*Opera med. Genev.*, 1736, Tom. I. Sect. 1., Cap. V., p. 60) long ago made mention of mental derangements, which remained after attacks of intermittent fever, and which, under the employment of depleting treatment, soon passed into a state of imbecility. He was surprised that no one had made mention of an occurrence which he himself had so frequently met with.

in another case, however, in which there was simply an accumulation of pigment, the development of the paralysis was gradual.

That the deposit of pigment in the substance of the brain and the abnormal cerebral symptoms are connected with one another as cause and effect, is a probable supposition, which acquires the more weight from the fact, that in general there is an unmistakable correspondence between the intensity of the two. Hence, the earlier observers, and especially Planer, had no hesitation in attributing the cerebral symptoms to the occlusion of the capillaries with pigment.

I cannot give my unconditional support to this view, however plausible it may at first sight appear, because a close analysis of the observations, and a careful comparison of the anatomical lesions with the symptoms present during life, render the connection between the two, as cause and effect, in many cases, at least, doubtful. Extensive interruptions to the circulation, with capillary apoplexies of the cortical substance of the brain, indicating rupture of the vessels and capillary hæmorrhage, constitute unquestionably a sufficient anatomical basis for the explanation of the cerebral symptoms, but obstruction to the circulation of the blood is by no means always present, even when the color is dark. The greater part of the pigment passes freely through the capillaries, and may be found in large quantities in the veins. The dark contents of the capillaries are nowhere so distinctly marked as in the white ground of the substance of the brain; hence they always appear more remarkable in this place than in other organs, of which the parenchyma is naturally darker. Even when the circulation of the blood does become arrested here and there, this is not to be regarded as a sufficient cause of functional disturbances, because the numerous branches of the vascular network permit of collateral currents.

On comparing the *post-mortem* appearances with the symptoms during life, we find, on the one hand cases where, in spite of the dark coloring of the brain, no cerebral disturbances have been present, and on the other hand, cerebral disturbances without any deposit of pigment. The last statement I have found to hold good six times, out of 28 cases of *Intermittens cephalica.* The older observers, such as Lancisi, Senac, Bailly, and also Maillot and Haspel, have made similar observations. We cannot, therefore, doubt, that the cerebral disturbances of intermittent fever above described, may occur without melanæmia, and that, consequently, there must be other causes, which can give rise to them. Hitherto, we have been unable to detect these causes. The fact of the morbid symptoms intermitting appears to argue in favor of their being periodically generated in the system, and again disappearing. I have already called attention to the circumstance, that in melanæmia the extensive decomposition of the blood pours periodically into the circulation, not merely the debris of the morphological disintegration of this fluid, but also the products of its chemical metamorphosis, which elude microscopic examination. A closer study of these metamorphoses may bring us nearer to the source of the cerebral symptoms.

The third organ in which we observe abnormal conditions of structure and functions in melanæmia, is the kidneys. The larger pigment cells and scales which enter these organs along with the arterial blood, not unfrequently become impacted in the capillary coils of the Malpighian bodies, and by altering the pressure of the blood, give rise to derangements in the secretion of urine, which exercise a powerful influence over the further progress of the disease. Albuminuria makes its appearance

to an extent which varies with the quantity of pigment found in the kidneys. In the cases where the fever has presented a distinctly intermittent type, and where, as in quartan fever, the intermissions have been of considerable duration, we have observed during each paroxysm a great increase of the albuminous contents of the urine, and during the intermissions a marked diminution, or a complete disappearance of them.

The albuminuria is frequently simple, and then the process may last a long time without leading to intimate structural changes in the kidneys. But in other cases, fibrinous matter passes off in the urine along with the albumen; I have repeatedly observed fibrinous casts, including pigment masses and granules of the same nature as those found in the blood; I have, moreover, met with cases in which bloody urine was excreted. Complete suppression of the urinary secretion is of frequent occurrence. In cases where the secretion of albumen and fibrinous matter has lasted for a long time along with intermittent fever, or after its cessation, and ultimately has led to a fatal termination from exhaustion, alterations of comparative insignificance have been found in the kidneys. Numerous flat, scarred depressions have been observed on the outer surface of the organ, but no distinct granulations. In some cases there has been lardaceous degeneration.

With the exception of the liver, the brain, and the kidneys, in no other organ or tissue can any remarkable change of structure, or derangement of function, be observed. It is true that we can detect pigment throughout the entire capillary vascular system, so far as this is filled with blood, but accumulations of this pigment, and occlusion of the capillary vessels, are only wont to take place to any extent where the smallness of the capillaries, or the peculiar nature of their distribution, impedes the free motion of the pigment scales. The supposed production of dyspnœa and œdema of the lungs, from occlusion of the pulmonary capillaries, is not supported by actual results, at least, in the cases which have come under my own observation. I do not believe that the inflammatory swellings which I have observed in isolated cases, such as limited deposits in the parotid and in the muscular tissue of the heart, are the result of extensive occlusion of the vessels. Close examination of the deposits in the muscular tissue of the heart discloses no remarkable accumulations of pigment.

The peculiar color of the skin which is produced by the quantity of pigment in the blood contained in the vessels of the cutis, is of importance in diagnosis. In the slighter grades of melanæmia it is ash-colored; in the more intense forms it is dirty grayish-brown, and sometimes deep yellow-brown. In most cases, a few drops of blood, obtained by scarification of the skin, suffices to show numerous particles of pigment, under the microscope.

v. *Pigment-Liver an accompaniment of Malarious Fevers.*

The derangements just described are ushered in and accompanied by a fever, which may assume very various forms and types.

In most of the cases the fever is an intermittent, and usually a quotidian or double tertian,—more rarely a simple tertian, and less frequently a quartan. The fever does not, as a rule, make its appearance in the regular form with distinctly-marked stages, but the intermission is usually incomplete; in most cases, the elevation of temperature and the increased

frequency of pulse do not disappear completely, although the repeated attacks of rigors with subsequent aggravation of the symptoms indicate the true type of the fever. After two or three incomplete intermissions, followed by a paroxysm, the fever in most cases becomes continued; patients have been repeatedly brought to the Hospital in this condition as cases of typhus. As a general rule, the intermittent type of fever is less marked, the more prominent are the local, and particularly the cerebral, derangements.

The pulse varies very greatly in frequency. In the most severe forms it does not usually exceed 80 or 90, which is an important circumstance for distinguishing the affection from typhus; in a few cases only has the pulse reached 120 or 140. Sometimes the pulse abates in frequency upon the supervention of nervous symptoms.

The three stages of the intermittent are seldom very distinctly marked; the rigor stage is often entirely absent, and distinct critical discharges by the skin and urine do not usually occur. Twice I have met with paroxysms of unusual duration (48 and 60 hours). The severe symptoms which usually accompany this form of intermittent, as a rule, make their appearance along with the fever. Sometimes severe brain symptoms, which speedily prove fatal, are developed after only a few hours of vague indisposition, without any distinct febrile symptoms manifesting themselves.

In other cases, a simple intermittent fever has existed for weeks or months, when suddenly a severe fit comes on, which often terminates fatally in an instant. This last form has occurred frequently in relapses of apparently simple tertian or quartan fever.

The cases vary in their progress: there are some which prove fatal in a few hours or days, whilst others are protracted over months; the cases with head-symptoms are almost always acute, the remainder are as frequently chronic. Out of 51 cases which I have observed, there were 24 acute and 27 chronic.

Such are the most important derangements which are observed in the disease under consideration. They seldom all exist together; in most cases one or other of them occupies a prominent place, while the remainder are either absent or developed in a milder form. In this way there arise varieties of disease which exhibit a wide difference in their progress and results. Four forms may be distinguished:—

I. Cases with predominant brain-symptoms.

II. Cases in which the kidneys are pre-eminently implicated.

III. Cases with predominant derangements of the gastro-intestinal tract, and of the appertaining glands, but particularly of the liver.

IV. In a fourth group, we may include those forms in which the local derangements are not very conspicuous, and do not influence essentially the further progress of the disease, but where the anæmia and hydræmia, resulting from affection of the spleen, constitute the most important morbid conditions. The pigment contained in the blood is here of subordinate importance, inasmuch as its quantity and characters do not give rise to extensive lesions of the capillary circulation; it is productive of no injurious consequences, provided we succeed in checking the anæmia.

In order to give some idea of the frequency of the individual derangements, and of their fundamental anatomical lesions, I may annex a short analysis of 51 cases observed here in Breslau. The results are, of course, only of value as regards the epidemic of this place, and must by no means be generalized indiscriminately.

Of the 51 cases, 38 terminated fatally, and 13 in recovery.[1] Severe brain-symptoms, such as delirium, convulsions, coma, &c., occurred 28 times out of the 51 cases; in 7 of these cases, there was no deposit of pigment in the brain; in 2 cases there was hæmorrhage in the cerebral membranes along with the pigment; and there was 1 case of cysticercus cerebri.

Out of the 51 observations, albuminuria was found in 20 cases, in 2 of which there was hæmaturia, and in 5, suppression of urine; albuminuria occurred without any pigment in 4 cases, whereof 2 were instances of lardaceous degeneration of the kidneys; in 5 cases pigment could be detected where there had been no albumen in the urine, but the quantity was scanty.

In 17 of the 51 observations, there was profuse diarrhœa, and 5 of these were cases of dysentery; profuse intestinal hæmorrhage was seen three times. Jaundice was present in 11 cases, but was always slight; bile-pigment also was observed in the serous effusions of the pleural cavities, without any distinct coloring of the skin and urine.

In all the cases which terminated fatally, the liver contained a quantity of pigment; in 10 it appeared enlarged and congested, and in 8 atrophied; in 9 cases the cells contained much oil; lardaceous matter could be detected in 3 cases, but only in small quantity.

Except in one case, pigment was always found in the spleen; three times this organ was lardaceous; and in 30 cases its volume exceeded the usual limits.

In a treatise upon affections of the liver, the cases which come under the first and second groups of melanæmia require only a passing consideration. I annex a few examples, in order to show that the liver is involved at the same time as other parts; and those cases only will be given in detail where the disease of the liver, and the disturbances resulting therefrom, were conspicuous.

I. CASES WITH CEREBRAL DISTURBANCE.

Observation No. I.

Tertian Intermittent Fever of three months' duration, ultimately with Coma during the fit.—Pneumonia, passing on to Consolidation.—Sudden Death.

Pigment-Spleen and Liver.—Consolidation of the Lungs.—The Brain free from Pigment.

W. Klein, aged 65, locksmith, was admitted in an unconscious condition on the 7th of January, 1855. He had been suffering for three months from tertian fever, which latterly had taken on an irregular type, and, during the fits, was attended by loss of consciousness. On the 8th, he regained the use of his mental faculties. On examination, the spleen was found to be considerably enlarged; on the left side of the thorax

[1] Of the cases which ran a favorable course, only those are here included in which pigment could be detected in the blood. These figures do not express the ratio of mortality, because the examination of the blood was often omitted, where no severe symptoms called for it.

there was dulness and bronchial breathing, extending from the middle of the scapula as far as the base; there was no expectoration. Pulse 96. The patient stated that during a frost, eight days before, he had felt a shooting-pain in the left side, and had expectorated saffron-colored sputa.

The next attack of fever was cut short by bark, in combination with sal-ammoniac.

The bronchial breathing and dulness continued unchanged, and were accompanied by a slight expectoration of gray mucus. The pulse varied in frequency between 80 and 90; the appetite was normal, the bowels regular, and the sleep quiet; neither albuminuria nor œdema was present. There was considerable anæmia.

He was ordered the ammoniated sesquichloride of iron.

On the morning of the 21st, the patient ate his breakfast; and while sitting upon a stool, in order to have his bed made, he became unconscious, and died.

Autopsy, 24 hours after death.

Neither the membranes nor the substance of the brain contained much blood; there was nothing abnormal either in the color or the consistence of the brain. No pigment was found in the capillaries of the cortical substance.

The mucous membrane of the bronchi was pale; the right lung was dry, bloodless, and emphysematous; the left was very firmly adherent to the costal wall; it was diminished in size; its tissue was firm, and not friable; its cut surface was very slightly granular, and of a uniform pale-brown color; the bronchial tubes were somewhat enlarged, and their lining membrane reddened; the upper lobe contained but little blood.

The heart contained loosely-coagulated dark blood.

The mucous membrane of the stomach and intestinal canal was pale.

The spleen was enlarged by about one-third, and slightly wrinkled; its parenchyma was flabby, tenacious (*zähe*), and of a bluish-black color.

The liver was of normal size, its color was blackish-brown. Its consistence was normal.

The bile was thick and yellow.

The cortical portion of the kidneys was atrophied.

The bladder and prostate were normal.

Observation No. II.

Febrile Gastric Catarrh.—Vertigo.—Convulsions.—Coma.—Return of Consciousness.—Parotid Swelling.—Albuminuria.—Death from exhaustion.

Melanæmia.—Accumulation of Pigment in the Spleen, Liver, the Gray Matter of the Brain, and the Kidneys.

Rosine Hornig, the wife of a day-laborer, aged 61, came to the Hospital on the 22d of August, 1854, after having suffered for four days from loss of appetite, headache, and giddiness. On examination, there was found to be consolidation of the apices of both lungs; the tongue was coated gray; there was slight distention of the epigastrium, and moderate

enlargement of the spleen. The pulse varied between 80 and 90; there had been no rigors.

On the 24th, the patient became restless, complained of severe headache, and was, when in this state, suddenly seized with general convulsions, which, with short intervals, continued for about two hours, and was followed by complete loss of consciousness. On the 25th, the consciousness had not returned. Pulse 84. The urine drawn off with the catheter, contained a considerable quantity of albumen, but no fibrinous coagula; involuntary stools; great restlessness; much sighing and groaning, so that it became necessary to remove the patient to a separate ward.

Here, as we were told, she was tolerably quiet; her consciousness gradually returned, but her memory continued bad and she only gave slow and unsatisfactory answers. A few days after, an inflammatory exudation made its appearance at the angle of the lower jaw, in the areolar tissue surrounding the parotid. By poulticing, this became soft, burst and discharged a large quantity of putrid pus. The patient then became more and more collapsed, and died from exhaustion, without any return of the convulsions, or loss of consciousness.

Autopsy, Sept. 11th, 20 hours after death.

The bones of the cranium and the membranes of the brain were congested; the cortical portion of the brain appeared of a chocolate color, and formed a marked contrast to the white substance; this last was of proper consistence, and contained the normal amount of blood. The microscope showed that there was a large quantity of pigment included in the capillary vessels.

Gray tubercular matter was infiltrated through the apices of both lungs; lower down, their parenchyma was moderately congested, but free from deposit.

The blood in the heart was coagulated, and of a dark color. Numerous pigment cells and scales could be detected in it.

The spleen was enlarged by one-half, and was pulpy, soft, and of a dirty grayish-brown color; numerous brown and black pigment cells and granules, like those in the blood, were found in this organ.

The liver had sharp margins and a smooth surface; its parenchyma was of normal consistence and of a grayish-brown color; some of its secreting cells contained much oil. The bile was pale and contained no albumen. In the stomach there was the cicatrix of a deep ulcer; its mucous membrane was pale; as was also the mucous membrane of the small and large intestines.

The kidneys were apparently normal; but, on closer examination, a tolerably large quantity of pigment was found in the glomeruli, and uniniferous tubes.

Observation No. III.

Quotidian Fever.—Enlargement of the Spleen.—Coma.—Death.—Melanæmia.—Accumulation of Pigment in the Spleen, Liver, Kidneys, and cortical portion of the Brain.

Elizabeth Ermler, a tailor's widow, aged 45, came to the Hospital on the 13th September, 1854. For a week before, she had had daily attacks

of fever; but previous to this, she had enjoyed perfect health. Her statements, however, seemed scarcely deserving of credit, owing to loss of memory and deafness, which had lasted for eight days. She could not say whether the fever had been ushered in by a rigor-stage. The respiratory and circulating organs were normal; the pulse was 90, small, and soft; tongue dry; abdomen soft and flat; no diarrhœa. The spleen extended about an inch beyond the false ribs; no roseolar eruption. The color of the skin was pale grayish-yellow, but this tinge was not very striking; the temperature was elevated.

She was ordered muriatic acid with bark.

The state of unconsciousness rapidly increased into complete coma. The countenance became pale, and the pupils small. The temperature continued high; and the pulse varied from 99 to 100. The urine, which came away involuntarily, was not examined. Death took place on Sept. 18.

Autopsy, 20 hours after death.

The membranes of the brain were tolerably congested, and the sinuses contained small colorless coagula. The cortical substance of the brain was of a leaden hue, and there was a considerable quantity of pigment granules and scales in its capillaries; the white substance of the brain was of normal consistence. A small quantity of serum was contained in the sac of the arachnoid.

The mucous membrane of the air-passages was pale; dark obsolete tissue was found at the apices of both lungs, accompanied on the right side by enlargements of some of the bronchi, which contained a calcareous pulp; posteriorly and inferiorly, there was œdema.

The heart contained a small quantity of loosely coagulated blood, in which numerous pigment particles could be detected.

The spleen was enlarged by about one-third; it was flabby, and its surface was wrinkled; its tissue was soft and of a grayish-brown color.

There was a tight-lace mark upon both lobes of the liver; the surface of the organ was smooth, its parenchyma friable and chocolate-colored; from the lower surface of the left lobe there projected the sac of an echinococcus undergoing disintegration. The hepatic cells were loaded with brown pigment, and contained but little oil.

The mucous membrane of the stomach was tumid, and of a livid hue; in the small and large intestine it presented, at some places, a slaty-gray color.

The kidneys were apparently normal, and the glomeruli loaded with pigment. A small quantity of slightly albuminous urine was found in the urinary bladder. The genital organs presented nothing abnormal.

Observation No. IV.

Fever of an undecided type.—Delirium.—Coma.—Vomiting.—Death on the fifteenth day.—No albuminuria.

Pigment in the Blood, the Cortical portion of the Brain, the Liver, Kidneys and Spleen.—Spleen slightly enlarged.

Bertha Meissner, aged 38, sempstress, was admitted in an unconscious condition on September 11th, 1854. She had been ill for fourteen days;

and after suffering from delirium and great restlessness, she had since the beginning of the last week completely lost her consciousness. The patient was of a pale, dirty-yellow color; her head and extremities were cool; her eyes were coated over with mucus; the pupils were of medium size, and acted somewhat slowly. The pulse was 84, and small; no enlargement of the spleen could be made out. The abdomen was tender; thin deep-brown stools were passed involuntarily. The urine was free from albumen.

Notwithstanding the employment of stimulants, the patient became very rapidly collapsed; and 14 hours after her admission she was a corpse. I did not observe any attacks of rigor, nor were any noticed by the relatives.

Autopsy, 15 hours after death.

The inner surface of the skullcap was covered by a thin layer of bony growths; the dura mater was at some places firmly adherent; the pia mater was anæmic; there was a small quantity of clear serum effused beneath the arachnoid. The cortical substance of the brain was of a dark ash-gray color; its capillaries were filled with pigment granules. The white substance of the brain was anæmic and of normal consistence; the lateral ventricles were of the usual size.

The air-passages and the lungs presented little that was abnormal; the posterior and inferior portions of the lungs were hypostatic and œdematous; the deposit of pigment in them was considerable.

The heart, as well as the large vessels, was normal. The blood, both in the right and left ventricles, was rich in pigment.

The stomach had a somewhat thickened, slaty-gray mucous membrane, presenting a few ecchymoses upon the prominent folds. The mesenteric glands and the intestinal canal were healthy.

The spleen was slightly enlarged; it lay very far back; its parenchyma was very soft, and dirty grayish-brown; brown and black pigment in the form of scales and cells could be detected in it in abundance.

The liver was of normal size; its surface was smooth and its edges sharp; its parenchyma was soft, and of a dirty grayish-brown color; the secreting cells appeared pale; the capillaries were filled with pigment. The gall-bladder contained a quantity of thin mucous, pale bile.

The surface of the kidneys was smooth; their parenchyma was shrivelled and anæmic; the glomeruli contained pigment scales. The urinary bladder was quite empty.

The uterus was that of a virgin; and there were small corpora lutea in both ovaries.

OBSERVATION No. V.

Intermittent Fever with Convulsions and loss of Consciousness.—Type irregular.—No rigor stage.—Recovery by treatment with bark.

Carl Grund, age 28, was admitted on the 27th of October, 1854. His illness commenced fourteen days before, and was characterized by persistent headache, accompanied by noises in the ears, nausea, and great weakness and delirium; there was no evidence, however, of rigor, or of any of the other symptoms of intermittent fever. On the 27th of October, soon after admission, the patient was seized with an attack of convulsions, accompanied by loss of consciousness, which lasted about half-an-hour and

returned once more during the night. On the morning of the 28th, a considerable enlargement of the spleen was detected; pulse 78; great headache, but consciousness clear; the skin secreting actively; the urine throwing down a slight deposit, but containing no albumen; stools normal.

The increased frequency of the pulse and elevation of the temperature appeared at irregular intervals, at one time daily, and at another time every second day, and were accompanied by giddiness and severe headaches, passing into delirium; these symptoms gradually subsided with sweating. They were never ushered in by a rigor. During the intermissions, the patient's condition was but little deranged. On the 8th of November, he was ordered 3 grains of quinine every two hours; under the use of this, the headache, giddiness, and excitement of the vascular system ceased; but on the 16th they returned in a severe and regular tertian form, though still without any rigor stage. Bark was administered for a long time; and under its use the symptoms gradually disappeared.

These attacks left the patient in a very low state, and he required a regulated chalybeate treatment, before he could be dismissed from the Hospital cured, and free from any enlargement of the spleen.

Observation No. VI.

Intermittent Fever of an irregular type.—Two paroxysms of 48 hours' duration.—Furious delirium during the paroxysms.—Cure.

Heinr. K. Gürtler, aged 39, a year before had suffered from intermittent fever for several weeks, and at the end of July was seized with violent pains in the region of the spleen, accompanied by moderate enlargement of the organ. Along with this there was bronchial catarrh, without any fever. He was treated at the Polyclinique with muriate of ammonia, and afterwards with senega.

At noon on June 29th, a moderately severe rigor came on, lasting about an hour, and followed by heat of skin, violent headache and furious delirium. Towards evening, the patient became completely unconscious; pulse 130; the skin dropping with perspiration. Not until noon of July 1st, did the sweating and frequency of pulse abate, and the consciousness return. In the evening, the patient was free from fever.

He was ordered three grains of sulphate of quinine every two hours.

On the afternoon of the 5th, the temperature became elevated; pulse 92 and *bisferiens;* tongue dry; severe headache. This condition lasted until the 7th, when profuse sweating and sudamina made their appearance, the pulse falling to 70.

By the continued use of quinine the patient rapidly recovered, and on the 16th was able to leave the Hospital.

Observation No. VII.

Quotidian Intermittent with severe Vertigo.—Febris vertiginosa of Paccinotti.—Cure by Quinine.

Carl Forster, aged 30, since the middle of September, had suffered from a quotidian intermittent, associated with severe vertigo. The patient

could scarcely advance two steps without staggering; on attempting to walk across the room, he fell repeatedly, and was obliged to be led. The color of his skin was grayish-brown, like that of a mulatto; the spleen was enlarged; there was no albumen in the urine. The pulse became more frequent; neither headache, rigors, nor any of the other symptoms of fever were present; the appetite was unimpaired, and the stools normal.

The patient having been treated before admission into Hospital, by cupping and purgatives, without any benefit, recourse was now had to quinine, whereupon the giddiness rapidly diminished, and ere long entirely ceased.

In a few days, the patient was able to be discharged from the Hospital, there being no anæmia to necessitate a longer treatment.

II. CASES WITH PREDOMINANT AFFECTION OF THE KIDNEYS.

Observation No. VIII.

Quotidian Intermittent of four weeks' duration; Diarrhœa; Albuminuria and Hæmaturia; Sudden Stupor; Convulsions; Death.

Accumulation of Pigment in the Spleen, Liver, Kidneys, and Brain.

C. Runschke, aged 50, came to the Clinique on the 3d August, 1854. For four weeks he had suffered from a quotidian fever, which had latterly become complicated with diarrhœa. His complexion was of an extremely pale grayish-yellow hue; there was no œdema, however, anywhere. The spleen extended an inch beyond the margin of the false ribs; the abdomen was soft and painless, and there was no ascites; the liver was of normal size; there was nothing abnormal in the respiratory organs or in the heart.

The urine was deep reddish-brown, and coagulated upon boiling, or on the addition of nitric acid. The pulse was 80; and there was much complaint of headache.

Was ordered quinine with opium.

On the 4th of August, the patient stated that he had an attack of rigors during the night; but the nurse had not observed it. The pulse was unchanged, 80, small and weak. The headache was considerably increased. One formed stool.

Ordered quinine with elix. acid. Hall.[1]

In the night, sudden restlessness and loud groaning. Professor Rühle, who was summoned to the patient, found him in state of deep coma, with irregular, interrupted respirations, and a small frequent pulse; the pupils were not enlarged; the temperature of the head was increased.

At 7 in the morning he expired; death being preceded by slight convulsions.

Autopsy, 14 hours after death.

The pia mater congested; the dura mater thickened and adherent to the inner surface of the skullcap.

[1] *Elixir acidum Halleri*, or *Mistura acida sulphurica*, is composed of concentrated sulphuric acid (1 part), and rectified spirit of wine (3 parts). Its specific gravity varies from 1005 to 1010. Dose 5 to 20 minims.—Transl.

After removing the meninges, the gray substance of the brain appeared of a dark chocolate color, and was defined by a sharp line from the central white matter. This copious deposit of pigment was very marked in the corpus striatum and cerebellum, and also in the pons Varolii. The white substance was intersected by fine blackish lines; besides this, the brain presented nothing abnormal in consistence, or in any other respect.

The air-passages pale; the lungs congested and œdematous.

In the heart there was a moderate quantity of loosely coagulated dark blood, with much pigment in various forms.

Spleen large, soft, and darkly speckled.

The liver normal in size; surface smooth; margins sharp; its parenchyma loaded with grayish brown pigment; the bile pale and copious.

The mucous membrane of the stomach was of a slaty-gray color; the lining membrane of the intestine was perfectly normal. The pancreas contained a larger quantity of pigment than is usual in such cases. The kidneys were lobulated, and, on section, presented a homogeneous, brown, glistening surface. There were numerous pigment cells and scales in the glomeruli of the Malpighian capsules.

The bladder contained a large quantity of bloody urine, and its walls were hypertrophied. There was a slight stricture of the urethra in front of the bulb.

Observation No. IX.

Continued Fever, with symptoms like those of Typhus.—Coma.—Albuminous and bloody urine containing clots loaded with black pigment.—Right Pneumonia.—Abortion.—Death on the sixteenth day.

Spleen and Liver soft and loaded with pigment.—Occlusion of the tubuli and uriniferi by pigment.

Rosalie Hellmann, aged 28, was admitted on August 25th, 1854, after having been treated for fourteen days, by a physician outside the Hospital, for a disease which was considered to be typhus. The woman was perfectly unconscious; her yellowish-gray skin was covered with perspiration; pulse 120, small and weak; respiration frequent, irregular, and stertorous. The thorax was of normal resonance in front, at some places slightly tympanitic; there was dulness at the lower and back part of the right side. Here bronchial breathing was audible; loud râles were to be heard in front; heart's sounds normal. The patient was in the eighth month of pregnancy.

At 10 in the evening, labor-pains came on, and about 2 in the morning a living child was born. The patient's general state did not improve. The dyspnœa and frequency of the pulse increased; the pulse rose to 136. The skin was warm and trickling with perspiration. The urine drawn off by catheter was bloody, and contained a quantity of albumen and fibrinous coagula. Death took place at 1 P.M.

Autopsy, 16 *hours after death.*

The membranes and substance of the brain were somewhat congested; the consistence and color of the latter were normal. The mucous mem-

brane of the air-passages was injected and covered with frothy mucus. The upper lobes of both lungs were congested and œdematous; the lower portion of the right lung contained no air, was friable and grayish-yellow, and exuded a purulent fluid upon pressure. The margin of the lower lobe of the left lung was collapsed. The heart was flabby and shrivelled; its valves were normal; the blood in the auricles was loosely coagulated, and contained much pigment.

The spleen was considerably enlarged, pultaceous and chocolate-colored. The blood of the splenic vein contained much pigment.

The liver was dark-brown, without any distinct indication of lobules; its cut surface was smooth, its margins sharp, and its consistence friable. The cells of the organ were very pale; the bile was dark, thick, and viscid.

The mucous membrane of the stomach was relaxed and injected, and covered here and there with ecchymoses. The mesenteric glands were small and contained no deposit; the mucous membrane of the small intestine was pale, without any abnormal development of its glandular apparatus; the large intestine was intensely injected.

The kidneys were of normal size; their outer surface was smooth, and they contained a moderate amount of blood. The glomeruli of the Malpighian capsules contained numerous brown and black pigment scales.

In the urinary bladder was a small quantity of bloody urine, containing small, pale, cylindrical clots infiltrated with pigment scales.

Observation No. X.

Quartan Intermittent Fever.—Intermittent Albuminuria.—Anasarca.—Dysentery.—Rapid cure by means of Quinine and Iron.

Henriette Schadek, aged 27, wife of a day-laborer, six months gone with child, came to the Hospital on the 8th of November, suffering from a quartan fever of six weeks' duration. There was much œdema of the upper and lower extremities, and also of the face; the urine was scanty and contained a quantity of albumen, and a number of fibrinous coagula, in some of which black pigment was observed. The spleen extended 4 centimètres ($1\frac{3}{5}$ inch) beyond the margin of the false ribs. The thoracic viscera was normal. It was stated, that the anasarca had made its appearance soon after the commencement of the fever.

The quantity of albumen contained in the urine diminished considerably on the 9th; and on the 10th it almost completely disappeared.

On the 11th a severe paroxysm set in, whereupon the albuminuria returned with renewed severity.

On the 13th there were frequent mucous stools mixed with blood, and accompanied by tenesmus. The fever was cut short by quinine; enemata of tannin and opium were administered to counteract the dysentery.

On the 18th the dysentery ceased, and the albuminuria disappeared.

On the 19th recourse was had to the lactate of iron; the quantity of urine became remarkably increased, and the œdema disappeared; so that on the 8th of December the patient was able to be discharged from the Hospital cured.

III. CASES WITH PREDOMINANT IMPLICATION OF THE LIVER AND GASTRO-INTESTINAL CANAL.

The implication of the liver is distinguished in many cases by a tenderness upon pressure over the right hypochondrium, as well as by an increase in the volume of the gland. These signs, however, may be absent even when the terminal ramifications of the portal vein are filled with pigment. We frequently observe a slight jaundiced tint of the skin and conjunctiva, together with brown bile, or allied coloring-matters in the urine; but these symptoms are not constant.

When the passage of the blood of the portal vein through the liver is interrupted to a great extent, we soon perceive the consequences of obstructive congestion upon the gastro-intestinal mucous membrane and the peritoneum; hæmorrhages take place, or increased secretion from the bowels, profuse diarrhœa, and sometimes also acute ascites supervene.

Intestinal hæmorrhages I have observed on three occasions; the bleedings were intermittent, and came on each time with the paroxysm of the fever; they were not affected by any treatment directed against the hæmorrhage, but yielded to large doses of quinine.

The first case of this nature which I met with, terminated fatally; because, from the continued character of the fever, the disease was supposed to be typhus. The case was that of a young man, aged 20, in whom severe intestinal hæmorrhage took place after he had been suffering for fourteen days from what his medical attendants called a slight attack of typhus. The patient, when I saw him, had passed several pounds of dark blood, and was much exhausted. His skin was grayish-yellow; pulse 110, scarcely perceptible. There were, first, three attacks of hæmorrhage, each relapse being preceded by an interval of two days; the attacks took place amidst great excitement of the vascular system, and were apparently arrested by styptics, such as alum, and the muriate of iron. The fourth attack, which also came on after an interval of two days, proved fatal.

Soon after this, I saw another patient at the same place, in whose case, likewise, the diagnosis of mild typhus had been made, from the fact that, after four attacks of a quotidian fever, the intermission had become indistinct; in this case also there was profuse intestinal hæmorrhage, which was subject to daily exacerbations, coming on always about the same hour. Styptics were tried in vain. Under the use of quinine, with elix. acid. Hall.,[1] the hæmorrhage ceased, and the patient became convalescent.

In a third patient with quartan fever, the intestinal hæmorrhage came on every three days, and was accompanied each time by hæmaturia; here, also, quinine sufficed to effect its removal.

Many cases of a similar nature occurred about the same time; I only mention those which I was able to observe most carefully. I have frequently found, upon *post-mortem* examination, dark suffusions upon the serous covering of the bowels and in the mesentery, resulting from the obstructions to the circulation. It could not be ascertained whether or not these cases were accompanied by intestinal hæmorrhage during life.

In other cases, where the obstruction to the circulation in the portal vein appeared to be less intense, there were profuse secretions from the intestinal mucous membrane, and rapid effusions of serum into the abdominal cavity. Diarrhœa occurred chiefly in individuals who were employed

[1] See note, p. 21.

in the water-works during the inundation of 1854; not unfrequently it passed into dysentery. I am, however, unable to state whether in these cases the interruption to the portal circulation was the only cause, or whether there were any additional causes in operation. Spontaneous catarrh of the bowels, unaccompanied by intermittent fever, was at the time rarely met with.

In cases where the effects of the accumulation of pigment in the portal vein were less marked during the persistence of the intermittent fever, more remote consequences might be observed at a later period. After the obliteration of a portion of the capillaries, the cells of the parenchyma in their neighborhood disappeared, and there arose a chronic atrophy of the liver, such as has been described in Chap. VI., Observations XXIV. and XXV., Vol. I., pp. 182, 183.[1]

Observation No. XI.

Persistent and oft-recurring Intermittent Fever, ultimately of a quartan type; Albuminuria of a high grade; fibrinous casts, with pigment in urine.—Œdema.—Rapidly-supervening ascites.—Tapping after the inefficacious employment of steel and purgatives.—Return of the Fever.—Paroxysm of two days' duration.—Death from exhaustion.

Pigment in the Spleen.—Occlusion of the hepatic capillaries.—Atrophy of the Liver.—Accumulation of Pigment in the Kidneys.—Consecutive Pneumonia.

Doroth. Schirmer, aged 38, a laborer's wife, was in the clinical ward from the 17th May to the 31st July. She had suffered repeatedly from intermittent fever of various types, and, during the last winter, from an almost uninterrupted quartan fever. At the time of her admission, the form of the fever was double quartan. For fourteen days, the patient had observed œdema of the feet, and a remarkable diminution in the secretion of urine; the urine was turbid, grayish-yellow, and contained an enormous number of fibrinous casts, which were partly covered by black pigment granules and cells. By boiling, the urine was converted into a solid coagulum. The region of the kidneys was tender upon pressure. The spleen extended about 4 centimètres (1⅝ inch) beyond the margin of the false ribs. The blood drawn off by means of a cupping-glass (care being taken to avoid the admixture of any foreign matter), contained brownish and black pigment masses, in the form of scales (*Schollen*) and cells.

After taking a drachm of quinine, the attacks of fever ceased. The characters of the urine, however, did not alter, and it continued scanty; there was repeated vomiting of greenish mucous fluid, without any headache or darkening of the countenance. Was ordered lemon-juice. The quantity of urine diminished somewhat, the vomiting ceased, and the appetite improved. Was ordered lactate of iron. The œdema of the feet diminished; but ascites became rapidly developed to such a degree as to interfere with respiration. Colocynth, gamboge, and similar purgatives

[1] Haspel (*Malad. de l'Algérie*, T. I., p. 335) appears to have previously observed a case of this nature in an individual who had suffered from obstinate diarrhœa, with ascites, after an attack of tertian fever, and whom it had been necessary to tap repeatedly; he found the liver small, not easily lacerable, and blackish internally.

were of little avail against the dropsy; the energetic employment of these remedies only gave rise to derangements of digestion, vomiting, &c., so that it did not appear advisable to persist in them.

On the 10th of July, twelve pounds of clear serum were drawn off by tapping. The volume of the liver, which could now be determined with greater accuracy, was slightly diminished.

On the afternoon of July 12th, there was an attack of fever,—rigors, heat, and sweating; the fever did not return on the 13th.

On the night of the 14th, after the usual interval, notwithstanding the employment of quinine, the paroxysm again made its appearance; the pulse continued very frequent, rose to 140, and was scarcely perceptible, without our being able to detect any cerebral disturbance, dyspnœa, or physical changes in the heart or pericardium. The pulse continued frequent from the evening of the 14th to the morning of the 17th, when it suddenly sank to 88. No fresh attack of fever took place; the pulse remained to the last varying between 80 and 90; but the ascites rapidly increased, and the œdema of the feet spread to a remarkable extent. The patient became rapidly prostrated; and, after a protracted agony, death took place on the 31st, without any cerebral symptoms.

Autopsy, August 1st, 12 hours after death.

No remarkable alteration was observed in the skullcap, meninges, or substance of the brain, either in their amount of blood, color, or consistence. The mucous membrane of the air-passages was pale; the left lung was œdematous superiorly; inferiorly, it was infiltrated, but only to a slight extent, with soft exudation; the pleura at this place was covered with a thin layer of gray flaky exudation. The right lung was hypostatic posteriorly, and emphysematous along its anterior margin. The pericardium contained about three ounces of clear serum; the muscular tissue and valvular apparatus of the heart were normal. The blood in the right auricle was firmly coagulated.

The œsophagus was pale. The mucous membrane of the stomach was ecchymosed at some places, and, around the pylorus, was of a slaty-gray hue. The lining-membrane of the bowel was pale superiorly, and in the large intestine was œdematous, and at some places the larger vessels were injected. The fæces were yellow. The pancreas and mesenteric glands were normal.

The spleen was not enlarged, but flabby and shrivelled; its capsule was thickened; and its parenchyma was tenacious (*zähe*), and of a bluish-gray color.

The left lobe of the liver had a broad atrophied rim; white traces of obliterated vessels could be seen in its serous covering. The parenchyma had a smooth surface on section, was somewhat friable, and of a grayish-brown color. The entire volume of the organ was diminished. The gall-bladder was found to be adherent to the stomach and colon, and was greatly distended with a green, mucous, somewhat albuminous bile.

On microscopic examination, the usual forms of black pigment could be detected in the spleen, in the blood of the portal vein, and in the capillaries of the liver.

The kidneys were of normal size; their surface was smooth; their capsule firmly adherent; and their cortical substance grayish-yellow, soft, and friable. The microscope showed the existence of pigment in the glomeruli,

as also in the vessels of the cortical substance, and occasionally, also, in the interior of the uriniferous tubes.

The mucous membrane of the bladder was ecchymosed; the urine was scanty and albuminous.

The genital organs were not altered to any great extent; there was a cyst the size of a hazel-nut in the right ovary.

Observation No. XII.

Intermittent Fever of a Tertian and Quotidian Type, lasting for seven weeks.—Intestinal Catarrh.—Hydræmia.—Anasarca.—Improvement under preparations of steel.—Relapse.—Rapid increase of the Dropsy. —Unconsciousness.—Death.

Pigment in the Spleen and Liver, without any implication of the Brain or Kidneys.

Franz Krocker, aged 45, a worker on the roads, came to the Hospital on the 1st of November, 1854. For seven weeks, he had suffered from an intermittent fever, at one time of a tertian, and at another, of a quotidian type; and, four weeks before, anasarca had made its appearance. The spleen was found to be considerably enlarged, extending about three inches beyond the margin of the false ribs; the heart and lungs were normal; there was no albumen in the urine; the bowels were relaxed and the motions thin.

After the arrest of the fever by means of quinine, the patient's condition improved; under the use of nutritious diet, wine, and the preparations of iron, the anasarca receded and the diarrhœa abated until the 8th of November, when the temperature became elevated, the pulse rose to from 96 to 100, and dyspnœa, with sibilus and rhonchus in the air-passages, and acute headache, made their appearance. At the same time, a copious effusion of fluid was detected in the abdominal cavity, and soon afterwards, in the pleural sacs likewise. Although this change in the patient's condition did not in any way resemble a fever paroxysm, yet considering the uncertain character presented by the malignant forms of fever, recourse was had to quinine. The patient, however, rapidly collapsed; his color became earthy; somnolence set in, and on the 14th he died.

Autopsy, 24 hours after death.

The body was greatly swollen from dropsy. The meninges and substance of the brain contained the normal amount of blood. There was no alteration in the color or consistence of the brain, and no pigment in the capillaries of the cortical substance.

The mucous membrane of the air-passages was slightly reddened, and covered with gray mucus. Several pounds of serum were found in each pleural cavity. The lungs were anæmic, and of a dirty-gray color, with emphysematous margins superiorly; inferiorly, they were compressed and non-crepitant.

The pericardium contained a pound of clear serum; in the heart, there was loosely coagulated blood, in which pigment was found in tolerable quantity. The muscular tissue and valvular apparatus of the heart were normal.

The peritoneal sac contained many pounds of clear serum. The stomach and intestinal canal were pale, but in other respects healthy.

The spleen was very large; its capsule was tensely distended; its parenchyma was soft and of a dark grayish-brown color.

The liver was shrivelled up at its margins; the serous membrane along the anterior rim of the left, as well as of the right, lobe, was opaque from recent exudation, and covered with pale-red newly-formed vessels. The hepatic tissue was of a dark grayish-brown color, and of soft consistence. Here, as well as in the spleen, there were large masses of pigment.

Bile, thin, pale, and albuminous.

Kidneys anæmic.

Urinary bladder and prostate normal.

Observation No. XIII.

Slight Dysentery.—Albuminuria.—Death from exhaustion.—No cerebral disturbance.

Accumulation of Pigment in the Spleen, Liver, Brain, Kidneys, and Pancreas.

Josepha Weiss, aged 54, wife of a day-laborer, came into the Hospital, on August 29th, 1854.

This woman was in a very low state, emaciated and anæmic, and for three weeks had been suffering from diarrhœa, accompanied by the passage of bloody mucus and great tenesmus. She referred this diarrhœa to a cold; she had not noticed any attacks of intermittent fever. On admission, the sphincter ani was so relaxed, that bloody mucous stools constantly flowed away from it; the urinary bladder was paralyzed; the dark urine drawn off by catheter contained a large quantity of albumen.

A transient improvement was obtained under the use of a suitable nutritious diet, wine, and enemata of decoction of calumba and tannin. Soon, however, the dysenteric affection increased. The exhaustion made rapid progress; the patient became more and more indifferent, slept much, but always gave perfectly intelligible answers. After a protracted agony, death took place on the 9th of September.

Autopsy, 26 hours after death.

The meninges were moderately congested; there was slight œdema of the arachnoid. The cortical substance of the brain was of a slaty-gray color, and sharply defined from the anæmic white matter.

The consistence was normal. The capillaries of the cortical substance were filled with granules and scales of pigment.

The lungs were dry and anæmic. There was much pigment in the blood contained in the heart.

The spleen was enlarged by about one-third, of a pulpy consistence, and dirty-gray color, and loaded with black and brown pigment.

The liver was of normal size, soft, and chocolate-colored, and its secreting cells were pale. The bile was scanty and dark, and contained no albumen. The capillaries of the liver were filled with pigment at the circumference of the lobules, and to a less extent near their centre. The

mucous membrane of the stomach was of a slaty-gray hue; the lining membrane of the small intestine was pale. The mucous membrane of the large intestine, from the ileo-colic valve as far as the sphincter ani, was covered with a gray puriform fluid; at many places it was abraded and marked by hæmorrhagic effusions, but it was free from any great amount of exudation, or from losses of substance of a deeper nature.

The pancreas was deeply tinged with pigment. The kidneys were anæmic and shrivelled (*welk*); the glomeruli, as well as the uriniferous tubes, contained a moderate amount of pigment.

There was much turbid urine in the urinary bladder, which, upon boiling, deposited albumen, but contained no casts of tubes. The genital organs were normal.

Observation No. XIV.

Abdominal Typhus.—Repeatedly recurrent Intermittent Fever.—Dysentery.—Exhaustion.—Death.

Spleen and Liver loaded with Pigment.—The latter organ atrophied.—Dysenteric ulcerations of the large Intestine.

E. Hahn, a female, aged 59, passed through a slight attack of typhus in September, after which she was seized with an oft-recurring intermittent fever. In the course of this, and without any other apparent cause, the digestion being tolerably good, and there being no diarrhœa or albuminuria, great anæmia came on within a short space of time: the skin assumed an eminently white appearance, and became dry and desquamating; extensive anasarca set in.

In November, several paroxysms of a quartan type occurred, which were soon removed by means of quinine, but which left behind a diarrhœa, gradually assuming all the characters of dysentery. Muriate of iron, nitrate of silver, with opium and other remedies, were employed to check this, without effect; the œdema rapidly increased; the patient passed into a state of collapse, and died from exhaustion on December 9th, death not being preceded by any loss of consciousness.

Autopsy, 12 hours after death.

The brain and its membranes, as well as the organs of respiration and of circulation, did not exhibit any important change, with the exception of paleness and an appearance of anæmia.

The spleen was of the normal size; its tissue was grayish-brown, and of moderate consistence. Its capsule was much wrinkled.

The liver was small; its outer surface was smooth, and its margins sharp; its parenchyma was moderately consistent and bluish-brown. The lobules were surrounded by dark rings. There was a small quantity of dark bile in the gall-bladder. The stomach and pancreas were normal.

The sigmoid flexure was drawn towards the right, and fixed there by bands of false membrane. The serous membrane of the rectum was opaque; its mucous membrane was very tumid, and covered with thick greenish-yellow masses of exudation; scattered over it were numerous patches of ulceration. These morbid changes extended, with a gradually

diminishing intensity, as far as the ileo-colic valve. The mucous membrane of the ileum was pale, and presented some gray typhus cicatrices.

The kidneys were rather small, at some places granular, but otherwise normal.

The right ovary contained a simple cyst, the size of one's fist.

VI. *Etiology.*

Considering the great frequency of intermittent fevers, the cases in which there is a marked development of pigment are comparatively rare; hence, in such cases other agencies, of which we possess no accurate information, must co-operate with the usual causes of intermittent fever. In the present defective state of our knowledge as to the nature of infectious diseases (*Infectionskrankheiten*), it cannot be determined whether a particular quality, or an unusual intensity of miasm is necessary for this purpose. The epidemic, to which the cases that I have described, belonged, made its appearance after an inundation in Silesia, resulting from an overflow of the Oder, in 1854. Since then, cases of this nature have very seldom been met with there, although the ordinary forms of intermittent fever are never absent.

VII. *Diagnosis.*

A perfectly accurate diagnosis can only be made by direct examination of the blood: a few drops carefully collected, so as to avoid contamination with any foreign matter, are sufficient to determine the presence or absence of large quantities of pigment. To the practised eye, the peculiar gray, ash-colored, or grayish-yellow color of the skin furnishes an obvious means of diagnosis. The supervention of severe cerebral symptoms with the coexistence of albuminuria or hæmaturia, or rapid collapse, is less to be depended on. More reliance is to be placed upon the occurrence of such cases in an epidemic form; this must especially guide us, when severe brain symptoms, intestinal hæmorrhages, suppression of urine, &c., are suddenly developed in the course of a fever of an indistinct type, without any other cause sufficient to account for these derangements. The periodic increase of such symptoms, the comparatively slow pulse, the increased volume of the spleen and liver, the color of the skin, &c., may supply further data for diagnosis. In some cases, the favorable effects of quinine may of themselves confirm the diagnosis.

VIII. *The Prognosis.*

The prognosis always remains a matter of doubt. The mere cessation of the fever does not entitle us to give a favorable prognosis, because sudden and unexpected relapses, speedily terminating in death, not unfrequently supervene. The cachexia and anæmia, resulting from the changes which the spleen and liver usually undergo, are ominous symptoms. The albuminuria, when it comes on periodically, and has not lasted a long time, yields readily to quinine; when of longer duration, it is often treated without any benefit. Coma and convulsions are in general of bad omen; yet such cases are not unfrequently cured by appropriate and well-timed remedies.

IX. *Treatment.*

The cure of the intermittent fever is the first thing to be done, and the necessity for this is the more urgent, the more severe the symptoms are during the paroxysm; every fresh paroxysm endangers life or increases the sum-total of the derangements which threaten to put an end to it. In such cases, so soon as we are sure of our diagnosis, large doses of quinine are sufficient, and the remedy is best given diluted with acids, in which form it is most easily and rapidly absorbed. The presence of counter indications must not be allowed to occasion delay, unless they are of a very important nature, such as great congestion of the brain.

In the slighter forms, with predominant abdominal derangements, great gastro-enteric catarrh, jaundice, congestive swelling of the liver, &c., it is judicious treatment to counteract these disturbances by suitable remedies, before having recourse to the quinine. This medicine must not be discontinued too early, because relapses are very apt to occur, and are particularly dangerous.

After the removal of the fever, the second indication is the treatment of the remaining local derangements of the spleen, liver, kidneys, brain, &c. Simple enlargements of the spleen usually yield to the use of quinine, and the easily absorbed preparations of steel, such as the hydrochlorate, the lactate, or the citrate of iron. It is a more difficult matter to effect the removal of the colloid infiltration of the organ, which is met with every now and then; for this purpose we must have recourse to the preparations of iodine, particularly the iodide of iron, and the mineral waters containing iodine and bromine, which are to be employed with more or less caution, according to the condition of the blood.

The congestion of the liver usually disappears spontaneously after the cessation of the fever, or even before this; when it remains stationary for a long period, we may employ rhubarb, the extractum saponariæ,[1] with the neutral salts, the ammoniated sesquichloride of iron, with extract of aloes. The same treatment is applicable in cases of enlargement of the liver, dependent upon biliary obstruction arising from catarrh of the duodenum and hepatic ducts.

A much greater danger, as regards the progress of convalescence, consists in the derangements of the nutrition of the liver, which are apt to result from the capillaries of the organ becoming loaded with pigment, and from the antecedent congestion conjoined with the alteration in the composition of the blood, produced by the marsh poison. What we have most to dread, is a gradually progressing atrophy of the gland, resulting from the destruction of numerous capillaries, the consequences of which we have already become acquainted with in Chapter VI., Vol. I., p. 174. I know of no treatment, by means of which such atrophy can be averted. We have already considered how the consequences of this atrophy, the gastric catarrh, the exhausting diarrhœa and the ascites are to be treated. The intermittent hæmorrhages and the acute ascites which succeed extensive obstruction of the capillaries, are best met by a speedy arrest of the intermittent fever; not until after this, can we have recourse to astringents and styptics.

The fatty and the colloid infiltrations of the liver, which occur in other cases, are to be treated according to the general principles laid down in subsequent chapters.

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[1] See note, Vol. I., page 220.

The third organ to be considered in the treatment is the kidneys; they become diseased, in such a manner as to excite apprehension, sometimes at an early date, at other times, not until a later period. Albuminuria and hæmaturia, which accompany the fever paroxysm, and remit and intermit along with it, yield best to quinine, and disappear for the most part, so soon as the fever ceases. When the abnormal condition of the secretion continues after the cessation of the fever, tonic and astringent remedies are suitable, such as the extract of cinchona, dissolved in some aromatic water, tannic or gallic acid, the preparations of iron, &c. A similar treatment, alternated with derivation to the skin and bowels, by means of warm baths on the one hand, and purgatives on the other (these last, however, only to be employed when the mucous membrane of the bowel is unaffected), is adapted to chronic albuminuria and the dropsy, which, in most cases, are developed, as the consequence of the cachexia remaining after the fever. The result in these cases depends chiefly on whether the affections of the spleen and kidneys are accompanied by still more important lesions of the liver, and of the mucous membrane of the stomach and bowels. When these parts are involved, it is seldom that we succeed in arresting the dropsy. When there is obstinate albuminuria, and at the same time a solid enlargement of the spleen, we have reason to believe that there is colloid infiltration of the kidneys, and we must employ the iodide of iron, so long as the condition of the digestive organs and the hydræmia will tolerate it; this is one of the most dangerous forms of the affection.

Cerebral disturbances during the febrile attack, demand a special treatment, when there is great plethora, or when there is a threatening of cerebral paralysis; in the former case, abstractions of blood, with cold applications, are to be employed; in the latter, diffusible stimulants, ether, musk, ammonia, &c. We must not, under these circumstances, neglect the use of quinine. Headache, giddiness, and other derangements, which remain after the removal of the intermittent fever, yield best to the protracted use of this alkaloid.

The altered composition of the blood, the anæmia and the hydræmia, in almost all cases, necessitate a tonic after-treatment, consisting in an easily-digested animal diet along with steel. The effects of this mode of treatment soon become apparent, when there are no important local affections in the liver, bowels, or kidneys, to interfere with assimilation, or to keep up abnormal secretions. When this is the case, all our endeavors to effect an improvement often prove fruitless.

CHAPTER II.

HYPERÆMIA OF THE LIVER, AND ITS CONSEQUENCES.

THE liver, in virtue of its structure and the arrangement of its vessels, is more subject than any other organ to abnormal distributions of its blood. The current of blood which, under the pressure of the heart, flows but slowly through the enormous and much ramified vascular network of this gland,—seeing that this pressure has already had to overcome one set of capillaries in the roots of the portal vein,—is acted on by agencies of a peculiar nature, as a result of which its rapidity and tension are liable to many variations.

Besides the heart's action, the organs in which the roots of the portal vein originate, influence the circulation in the liver. During digestion, when the abundant secretion of the mucous membrane of the stomach and bowels causes an active flow of blood to these parts, and when, at the same time, large quantities of fluid are absorbed, the blood flows more actively through the portal vein to the liver than is the case at other times.[1] Similar consequences may result from the irregular contractions of the splenic vessels, which are amply provided with muscular fibres.

The hepatic circulation is still more affected by the action of the diaphragm and abdominal muscles, by means of which the abdominal cavity is narrowed, the portal vein compressed, and its contents carried towards the constantly open vessels of the liver.[2] The contractions of the muscular tissue of the stomach and intestines produce a like effect upon the delicate roots of this vein, although in a more limited degree.

A third important agency affecting the circulation of the liver, is the varying opposition made to the escape of blood by the hepatic veins. This diminishes during each inspiration, and increases with the expiration. During the inspiration, the blood is drawn towards the heart from the open hepatic vessels, whilst at the same time the portal vein and the

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[1] According to Cl. Bernard, in order to protect the liver from too great a pressure of blood during the period of digestion, special communications exist between the portal vein and vena cava, which carry the blood directly into the vena cava, without passing through the portal capillaries.

[2] The earlier physicians had peculiar notions as to the circulation of blood in the portal vein, upon which they grounded their theories of obstructions and enlargements (*Anschoppungen*) of the liver. Boerhaave believed that the blood in the portal vein circulated independently of the heart. "Sanguis venæ portarum amittit omnem a corde acceptum impetum (*Prælect. academ.* Ed. Haller, vol. III., p. 115). The blood, he thought, was propelled through the liver by means of the muscular tissue, with which the portal vein is furnished in the capsule of Glisson (*ibid.* pp. 115 to 118), as well as by the respiratory movements of the diaphragm and abdominal muscles (*ibid.* p. 183). Stahl likewise denied the influence of the heart, and thought that the blood of the portal vein was propelled by the respiratory movements, and by a peculiar tonic motive power, having its seat in the spleen, intestines, mesentery, &c. Even at the present day, these views are brought to bear upon the doctrine of abdominal plethora.

liver are compressed by the descent of the diaphragm, and their vascular contents propelled further towards the heart. This movement is favored by the contraction of the greatly developed muscular tissue of the hepatic veins.

Besides these influences, which for the most part are due to the position of the liver and its vessels, and which are very variable in the extent of their operation, the hepatic circulation is influenced by other agencies, which are less accurately known. Among these may be mentioned the contractility of the walls of the vena porta, as well as of those of the hepatic vein and artery, which appears to be chiefly dependent upon innervation, and which, in rare cases, is liable to modifications from an altered state of their nutrition. When treating of chronic atrophy of the liver, we ascertained that dilatations of the portal vein may result from interruptions to the nutrition of the vascular walls; other forms of derangement arising from a similar cause will afterwards be brought forward. We know little that is positive as to the influence exerted by the nerves over the circulation of blood in the various divisions of the portal vein and in the hepatic artery. According to the experiments and observations of Cl. Bernard, injuries of certain localities of the medulla oblongata (*Diabetic puncture*), electric irritation of the proximal extremity of the divided vagus nerve, contusions of the head, poisoning with curari, &c., may give rise to hyperæmic swelling of the liver; I have myself observed a remarkable degree of congestion of this organ after division of the splanchnic nerves, and removal of the greater part of the cœliac ganglion. As might have been expected, the circulation of the blood in this organ is modified to a greater extent than we are aware of, by nervous influences. These still require investigation, because we are by no means entitled, without further investigations, to apply to the portal vein the results of experiments which have been made on other portions of the vascular system.[1]

From what has just been said, it will be understood, that in a system of vessels subject to such manifold modifying agencies, various derangements may occur, the causes of which are at one time obvious, but at another time less intelligible. We confine ourselves, in the first place, to the simpler forms, the etiology of which is more easily investigated, and shall afterwards pass on to the consideration of the more complicated varieties.

A. *Hyperæmia of the Liver, arising from obstruction to the circulation of the blood.* (*Stauungshyperämie.*)

1. *Pathology.*

In valvular diseases of the heart, and especially in such as give rise to accumulations of blood in the venæ cavæ at an early period, such as constriction of the left auriculo-ventricular opening, incompetence of the mi-

[1] Kölliker and Virchow observed in the body of an executed criminal, that no contraction resulted from galvanic irritation of the trunk of the portal vein, and only a slight contraction in the superior mesenteric vein. In experiments made by myself and Reichert on living dogs, a distinct, although slight, contraction was observed to take place in the splenic and mesenteric veins; in the trunk of the portal vein this was indistinct; no contraction could be observed in the hepatic veins and vena cava inferior, in which, from their very muscular character the effects might have been expected to have been more remarkable.

tral, and still more of the tricuspid valves, and further, in affections of the lungs, which greatly impede the circulation through the pulmonary artery, such as emphysema, extensive induration or atelectasis, in constriction of the thoracic cavity from angular curvature, in great pleuritic effusions, &c., hyperæmias of the liver are met with as a general rule. Under such circumstances, a powerful agency in the circulation of the blood in the liver, viz.: the suction power exercised during inspiration is either more or less powerful than it ought to be; either the blood in the vena cava and hepatic veins is subjected to a higher degree of pressure, which interferes with the evacuation of the capillaries of the portal vein, or the current of blood regurgitates with each systole of the heart into the hepatic veins, as may be made the subject of direct observation in the case of the veins of the neck.

When this is the case, the branches of the hepatic vein in the liver continue permanently distended, and gradually enlarge, while their walls become hypertrophied; the stagnation is propagated to the portal vein, and to the organs in which this vessel takes its origin; and there results a series of derangements in the functions and nutrition of the liver and of the organs comprised in the portal system.

The liver enlarges in every direction, without any important alteration in its form; its capsule becomes tightly distended, and the consistence of its parenchyma firmer.[1] On section, the organ presents a nutmeg-like appearance, which can also be seen through the capsule, and which varies in character according to the degree of the congestion. Usually there may be seen rounded, simple, or branched dark brownish-red patches, surrounded by portions of tissue of a pale-brown color. The dark spots correspond to the situations of the hepatic veins, and their form depends upon the direction in which the section of the vessel passes.[2] The light-colored portions of the parenchyma exhibit upon closer inspection, pale delicate ramifications corresponding to branches of the portal vein.

When the congestion attains a more advanced stage, the dark parts become incorporated together, and enclose in an annular manner portions of the pale tissue which corresponds to the branches of the portal vein; the forms of these pale parts then appear to be, for the most part, rounded or oval, sometimes they are elongated, or divided in a dichotomous manner, and more rarely they resemble a leaf.

In the highest grade of hepatic hyperæmia, the organ exhibits over extensive portions a uniformly dark-red color, in the midst of which the reddish-black outlines of the greatly distended branches of the hepatic vein can be made out[3]

The obstruction in the hepatic veins necessarily interferes with the circulation of the blood throughout the entire capillary system of the liver, and through this reacts upon the portal vein, as far as its primary roots. The secretion of the liver does not appear to suffer any important change:

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[1] Here and there, the parenchyma is doughy and œdematous, serum can be squeezed out of it, and it retains impressions made upon it with the finger.

[2] Even in the normal state, the blood is retained here in considerable quantity, and indications of such a nutmeg-appearance are not of very great importance, especially in cases where death has proceeded from disease of the lungs. For other causes of this appearance of the liver, see Vol. I., page 208, note [1].

[3] Kiernan (*Transactions of the Royal Society for* 1833) has correctly described this variation in the degree of hyperæmia of the liver, but the numerous delineations of these varieties given by him are far from natural.

I have not been able to convince myself that it is either increased or diminished; in a few cases, the bile contains albumen. The mucous membrane of the bile ducts are often seen to become swollen, and to throw out an increased secretion, and in this way the ducts themselves may become partially obstructed, and occasionally, also, a stoppage to the flow of bile, with slight jaundice may be observed. The nutrition of the hepatic cells remains at first unaffected, but after a time, we observe in those which are situated nearest the commencements of the hepatic veins, an abundant deposit of fat, such as in other cases is only met with in the cells next the branches of the portal vein.

From the persistence of this obstruction to the circulation of the blood, there is gradually developed a peculiar atrophy of the hepatic parenchyma, which formerly was frequently confounded with cirrhosis. The gland which hitherto has been swollen from the quantity of blood which it has contained, begins to diminish in size, and at the same time its surface, and afterwards its parenchyma, acquire a finely-granular structure. The granulations arise from the circumstance that the central veins of the lobules, and the capillaries opening into them, or in other words, the roots of the hepatic veins become enlarged under the powerful pressure of the obstructed blood, and thus lead to a disappearance of the hepatic cells enclosed in their meshes. The cells situated in the middle of the lobules become atrophied, and in their place appears a soft vascular tissue, consisting of enlarged capillaries and newly-formed areolar tissue, whilst the cells of the periphery of the lobules nearest the branches of the portal vein remain unaffected.[1] The farther this atrophy extends, the more does the entire volume of the liver diminish, and the more distinct do the granulations become.

Through the hepatic veins the obstruction is propagated to the portal vein, and to the structures in which the roots of this last vessel take their origin. The vessels of the mucous membrane of the stomach and intestine, of the spleen, pancreas, &c., become distended with venous blood; and, as a consequence of this, their nutrition and secreting powers are deranged. These results manifest themselves first and most distinctly in the mucous membrane of the stomach and bowels; this assumes a more or less dark-red color, its tissue becomes softened, infiltrated with serous fluid, and consequently swollen; while not unfrequently, extravasations of blood take place, which give rise sometimes to a deposit of black pigment, and sometimes to erosions and ulcerations. The secretion is usually increased in quantity and altered in quality, being thinner and more watery, or it often continues scanty. At the same time, watery effusions take place from the serous surfaces of the abdominal cavity. The spleen increases in volume, but only for a time, and never to a remarkable degree; at a later period it becomes more solid and firm, and is reduced to its normal dimensions.[2] In many cases, we find old extravasations of blood between the folds of the mesentery, the mesenteric glands of a livid tinge, with congestive swelling and serous infiltration of the pancreas. In consequence of the increased pressure on the part of the blood of the portal vein, the

[1] These appearances can be traced best in the injected liver.

[2] In thirteen cases of organic disease of the heart, in males, the average weight of the spleen was 0.23 kilogrammes (8.1 oz. avoird.); its relative weight to that of the body was as 1 to 276; of nine cases, in females, the absolute weight was 0.17 kilogr. (6 oz. avoird.), and the relative weight as 1 to 282; in thirteen cases, of females, in which there was also extensive dropsy, the absolute weight was 0.14 kilogr. (5 oz. avoird.), and the relative as 1 to 464. See Vol. I., pages 18 and 19.

absorption of the liquefied ingesta, so essential for nutrition, is greatly impeded.

It is worthy of notice, that, although the pressure must be uniformly distributed from the trunk of the portal vein over its radical branches, still the results of the obstruction are by no means uniform in the various parts from which these branches come. Of the gastro-intestinal mucous membrane, it is only some portions which are always seriously diseased, whilst the remainder appear but little changed. Disease of the mucous membrane of the stomach is the most constant, and the most marked; out of 20 cases, this membrane was of a dark-red color in 12, and in 7 it was covered with ecchymoses and erosions; in 4 cases only was the membrane relaxed and of a pale color. The small intestine is quite different from the stomach in this respect; as a general rule, the lining membrane of this portion of bowel is pale and scarcely altered; in 5 out of 20 cases only, was the ileum moderately congested, and never to such a degree as the stomach. The large intestine, and particularly the cæcum and sigmoid flexure, have been observed to be congested somewhat oftener, or in 7 out of 20 cases; this congestion has at one time been uniformly distributed over the greater extent of the mucous membrane; at other times, it has been limited more to the folds of the bowel, and to the circumference of the solitary glands; in no case did its intensity equal that of the injection of the stomach.

These inequalities in the distribution of the congestion appear to be dependent upon the differences presented by the individual portions of the intestinal canal, as regards their vascularity, and the size and position of their vessels; besides these causes, there are other circumstances, which in many cases must co-operate, such as the irritation of ingesta or medicines, and also the variations in the degree of *post-mortem* congestion, produced by the accumulation of gas in isolated portions of the bowel.

Independently of the portal system, we find the derangements just described almost always accompanied by albuminuria, which sometimes is persistent, and at other times is only observed from time to time. This morbid condition is of importance as regards the affection of the liver, inasmuch as all the derangements referrible to the portal vein, including the affection of the liver itself, are less prominently developed, the greater the loss of albumen by the urine, and the more marked the anæmic condition, which is gradually induced by this drain upon the system, as well as by the interference with digestion and intestinal absorption.

II. *Symptoms.*

The several stages in the development of mechanical hyperæmia of the liver, may usually be traced with ease at the bedside of the patient. The enlargement of the gland first makes itself known by a feeling of tightness and heaviness in the right hypochondrium, usually associated with more or less severe symptoms of gastric catarrh, and not unfrequently with slight jaundice. Percussion and palpation at once indicate an increase in the volume of the organ, the extent of which varies with the degree of obstruction to the circulation; during an attack of violent dyspnœa and cyanosis, the dulness soon increases to the extent of several centimètres. The surface of the organ at first feels smooth and tense; at a later period, it is uneven and granular. After a long continuance of the disease, the volume of the organ gradually diminishes; and this dim-

inution is more rapid the more complete is the obstruction to the circulation, and the earlier the date at which a marked degree of anæmia shows itself.

Besides the local changes in the liver itself, we observe the effects of the obstruction to the portal circulation, manifesting themselves chiefly by derangements[1] of digestion, pains and tension in the epigastrium, nausea, &c., and occasionally, also, by enlargement of the hæmorrhoidal veins. The bowels, as a rule, are confined; in rare cases, a transient diarrhœa makes its appearance (only in 4 out of 20 cases). The scanty, highly-colored urine, almost always contains small quantities of albumen, and sometimes also of bile-pigment; the latter of these complications is in most cases only temporary, and is associated with a slight jaundiced tinge of the conjunctiva and skin.

These derangements of the abdominal organs are accompanied by the symptoms of the cardiac or pulmonary disease, which constitutes the primary cause of the obstruction to the circulation, and which, sooner or later, leads to death from pulmonary œdema, apoplexy, general dropsy, &c.

III. *Treatment.*

In the treatment of the hepatic affection under consideration, we must keep in view, that it constitutes only a link in the long series of pathological processes, which are originated and kept up by one common cause, and that it depends upon the nature of this cause, whether the results produced are temporary or permanent. In most cases, palliative treatment is all that is possible, inasmuch as the fundamental organic lesions cannot be removed. The main point to be attended to is to reduce the congestion of the portal system, and to moderate the injurious consequences which this produces upon digestion and nutrition. A careful fulfilment of these indications contributes greatly to the relief of the complaints which are necessarily associated with cardiac diseases, and to ward off the dangers to which they give rise. Considerable swelling of the liver, accompanied by pain in the right hypochondrium, is most easily reduced by the mild saline purgatives, the action of which may be kept up for a longer period by infusion of rhubarb; at the same time, we may apply cupping-glasses over the hepatic region, and leeches to the anus. Where the obstruction to the circulation is not of too threatening a character, we may often succeed in procuring permanent relief by the careful employment of the Ragoczy Spring of Kissingen, or of the Mill Springs of Karlsbad. I have under my care a patient suffering from constriction of the left auriculo-ventricular opening, who for four years contrived to pass a tolerable winter by using the cooler springs of Karlsbad; and I have repeatedly obtained similar results by the careful employment of the waters of Marienbad and Kissingen.[2] In cases where the disease of the valves or of the muscular tissue of the heart is far advanced, the mineral waters can no longer be borne.

On the appearance of dropsy we must avoid saline medicines, and substitute for them the bitter vegetable substances, such as rhubarb,

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[1] Not unfrequently we find, upon *post-mortem* examination, all the signs of chronic catarrh of the stomach, tumefaction, lividity, a thick layer of mucus, &c., in cases where no indications of any derangement of digestion had existed during life.

[2] See note, Vol. I., pages 42 and 88.

aloes, and similar preparations, to which must be added, in the event of tympanites, aromatics, æthereal oils, and small quantities of ether. When there is great tenderness of the stomach, we must apply a few leeches, or confine ourselves to the employment of the watery tincture of rhubarb, with cherry-laurel water, extract of belladonna and similar remedies. Gastric hæmorrhage, which, however, is seldom profuse, and also ulceration of the stomach, call for the employment of ice and astringents.

We must beware of inflicting a lasting injury upon the digestive organs, by persisting in the use of digitalis.

Observation No. XV.

Constriction of the left auriculo-ventricular opening of the Heart.—Incompetence of the tricuspid valves.—Marked venous pulse.—Hæmoptysis.—Albuminuria.—Mechanical hyperæmia of the Liver, Stomach, and large intestine.

Carl Scholz, a journeyman baker, aged 38, was admitted into Hospital on December 19th, 1854, and died on the 19th February, 1855. Five years before, the patient had suffered from an attack of acute articular rheumatism, which had been followed by cardiac palpitations, dyspnœa, and bloody expectoration.

On admission, there was marked dyspnœa, extensive râles, and finely-frothy sputa tinged with blood. The impulse of the heart was felt at its normal site, and over the apex there could be heard a systolic and diastolic murmur; the systolic sound was heard still louder over the ensiform process of the sternum; both disappeared in an upward direction; the second sound of the pulmonary artery was unusually loud. The cardiac dulness was increased in width. The countenance, and particularly the lips, were of a cyanotic hue. The veins in the neck were greatly distended, and exhibited systolic pulsation in a marked degree. The pulse in the radial artery was 80 and small. The liver extended about 1½ inch beyond the margin of the ribs, the hepatic dulness in the mammary line amounting to 11 centimètres (4⅓ inches); its surface felt uneven and communicated a sensation of pulsation to the hand placed over it. The tongue was clean; the appetite slightly impaired; and the bowels confined. The urine was scanty, dark, and slightly albuminous.

Was ordered infusion of digitalis with 2 grains of extract of aloes, daily.

The dyspnœa and abdominal tension were relieved, but only for a time. Œdema of the feet and ascites gradually became developed, and there were repeated attacks of hæmorrhagic œdema of the lungs (*blutiges Lungenœdem*), which had to be removed by digitalis and benzoic acid. Up to the beginning of February, the patient continued tolerably comfortable, his appetite kept up, and his sleep was but little disturbed; but from this date, without any alteration in the auscultatory signs above described, the dyspnœa, cyanosis, and dropsy daily increased, and were accompanied by complete prostration of the digestive powers, until on the 19th the patient had an attack of asphyxia, which terminated in death.

Autopsy, 17 hours after death.

The cerebral membranes were congested and thickened; the substance of the brain was normal. In the neck was observed a remarkable enlargement of the internal jugular vein, which presented a bulbous projection above the clavicle, the size of a walnut; the external jugular vein was likewise enlarged.

The mucous membrane of the air-passages was congested, and at some places suffused with blood; both lungs were crepitant throughout, much congested, but not œdematous. The pericardium was opaque white, and thickened; over the right ventricle, which at its base measured 3¼ inches in breadth, was a large fibrous spot (*Sehnenfleck*); the tricuspid valve was thickened and opaque, its margins were rounded; the chordæ tendineæ, as well as the flaps of the valves, were adherent to one another; in consequence of this the auriculo-ventricular opening was much enlarged, and its valvular apparatus incompetent. The endocardium of the enlarged auricle had an opaque white appearance, the foramen ovale was one line in diameter: the valves of the pulmonary artery were normal. The walls of the left ventricle were unaltered; the bicuspid (mitral) valve was connected to a rigid ring, and at its base were rough calcareous deposits; the coronary veins were much enlarged; the aorta was normal.

The mucous membrane of the stomach was intensely congested, bluish-black, and at some places covered with gray flakes; the lining membrane of the jejunum and ileum was pale; the cæcum and colon were at some places congested and ecchymosed. The mesenteric glands and veins were unaltered.

The spleen was of normal size; its capsule was thickened; and its parenchyma reddish-brown, firm, and congested.

The liver was large, its capsule opaque, and its surface unevenly granular; its parenchyma was very congested; its color appeared at some places of a uniform dark-red, with black specks corresponding to the hepatic veins; here and there spots could be distinguished, partly white and partly of a greenish-yellow color, surrounded by dark congested spaces. The vena cava descendens and the hepatic veins were enormously enlarged, and their walls were thickened. This enlarged condition extended, as was shown by injection, as far as the capillaries of the lobules; the hepatic cells were partly atrophied, and partly filled with oil globules; in the greenish-yellow spaces the cells were loaded with bile-pigment. The gall-bladder contained a scanty, thick, dark secretion.

The cortical substance of the kidneys presented a few isolated superficial scars.

Observation No. XVI.

Constriction of the Left Auriculo-ventricular opening.—Repeated Attacks of œdema of the Lungs.—Albuminuria.—Congestion of the Liver from Obstructed Circulation.—Hæmorrhagic erosions of the Stomach.—Intestinal mucous membrane, pale and tumid.

Veronica Gräser, a joiner's wife, aged 29, was admitted on October 31st, 1854, and died on the 6th of December following. She had already, three years before, been treated in the Hospital for œdema of the lungs, which became developed soon after delivery, in consequence of con-

striction of the left auriculo-ventricular opening of the heart. Even then the same physical signs could be made out, as on the patient's last admission. A diastolic murmur, associated with *frémissement*, could be heard loudest over the apex of the heart, which was felt to beat at its normal place; this was accompanied by a remarkable exaggeration of the second sound to the left of the sternum in the second intercostal space; the cardiac dulness was increased in its transverse diameter, and extended beyond the right margin of the sternum. The pulse was 90, small and irregular. There was very extensive bronchial catarrh, a moderate amount of effusion in the right pleural sac and in the abdominal cavity, and œdema of the feet. The renal secretion was scanty; and there was a small quantity of albumen in the urine. The liver extended about 2½ inches beyond the margin of the ribs; its dulness in the mammary line amounted to 14 centimètres (5½ inches); the surface of the gland was smooth, its margin sharp, and its consistence firm. There was no appetite, although the tongue was clean; the bowels were somewhat confined, and the stools of a dark-brown color.

Was ordered infusion of digitalis leaves with tincture of rhubarb and acetate of potash.

The respiration became freer, and there was an increased secretion from the kidneys and the bowels; the œdema of the feet disappeared; the hepatic dulness diminished by about 3½ centimètres (1.378 inch).

Up to the 18th of November, the patient continued to improve under the use of a weak infusion of rhubarb; the digestive powers rallied; the bowels were moved regularly; and the dyspnœa was less violent. On the 18th, the pulse became very irregular; the dyspnœa again increased; there were finely-frothy sputa; the volume of the liver attained its former size. Under the use of the ethereal tincture of digitalis, the rhythm of the heart's action certainly improved; but the nausea, which soon supervened, and the complete loss of appetite, rendered it necessary to exchange this preparation for mild bitter remedies. The urine was always scanty, and its albuminous contents inconsiderable; the ascites and œdema of the feet rapidly increased.

On the 5th of December, there was repeated vomiting, great debility, imperceptible pulse, and œdema of the lungs. Death on December 6th.

Autopsy, 25 hours after death.

The brain and cerebral membranes anæmic, but otherwise normal. In the right pleural sac were found about three pounds of clear serum; the surface of the left lung was everywhere firmly adherent. The mucous membrane of the air-passages was of a bright-red color, and covered with small ecchymoses. The parenchyma of the lungs was dark-brown, œdematous, and somewhat firm in consistence; the lower lobe of the right lung was compressed; in the middle lobe was found a hæmorrhagic infarction the size of a walnut.

There were about seven ounces of serous fluid in the pericardium; the heart was considerably enlarged transversely; its right ventricle was dilated and hypertrophied, the hypertrophy being particularly marked in the *conus arteriosus;* the valves were normal; the left auricle was enlarged, and its muscular tissue thickened; the left auriculo-ventricular opening appeared narrowed to an oval slit, scarcely admitting the point of the forefinger; the margins of the valves, and a portion of the chordæ tendineæ

adhered to a smooth thick ring. The muscular tissue of the left ventricle, and also the aorta, were normal.

There were about seven pounds of fluid in the abdominal cavity. The mucous membrane of the stomach, which was covered by a tenacious blackish substance, presented hæmorrhagic erosions, which were most numerous in the neighborhood of the pylorus; its tissue was everywhere tumid and congested. The mucous membrane of the intestine, however, was throughout pale and relaxed.

The spleen was of normal size, dark-brown, and firm.

The liver was enlarged, its surface was slightly granular, and its margins sharp. The gall-bladder contained a considerable quantity of thin bile, in which albumen was detected.

The left kidney was of normal size; small depressed scars were observed upon its surface, and its parenchyma was somewhat firmer than usual. The right kidney was much contracted; its surface was scarred and furrowed; its cortical substance had disappeared; a yellow infarctus the size of a sechser (or of a threepenny-piece) was observed upon its convex edge.

No important pathological change was found in the urinary bladder or genital organs.

In addition to the morbid conditions of the heart and lungs, which, from the fact that the entire mass of the blood must pass through these organs, pre-eminently give rise to obstructions in this portion of the venous circulation, we observe, although with comparatively less frequency, mechanical congestions of the liver, produced by constrictions of the vena cava inferior above the opening of the hepatic veins, as also by constrictions of the hepatic veins themselves at their entrance into the vena cava. Watson[1] has described an instance of hyperæmic swelling of the liver, extending as low down as the crest of the ilium, which was caused by the lateral pressure upon the vena cava of an aortic aneurism. After the bursting of the aneurism, the pressure upon the vena cava ceased, the stagnating blood flowed on, and the organ had returned to nearly its normal size before the *post-mortem* examination was made. I have myself observed slight obstructions result from the compression of the vena cava by retro-peritoneal cancerous tumors. I have also seen a constriction of the hepatic veins caused by valvular projections into the interior of this vessel in a case of cirrhosis of the liver; in this case, the obstruction had given rise to numerous apoplectic patches in the parenchyma of the liver.

However obvious the effects of the obstruction may be, it is seldom that we can arrive at a perfect knowledge of derangements of this nature during the life of the patient. This remark applies also to the partial mechanical hyperæmias, which are developed as a result of deep fissures in the liver from tight-lacing; and to the swellings of one lobe caused by induration of the other, or by obliteration of one of the branches of the portal vein, whereof I have before me several observations.

B. *Congestions and Atonic Hyperæmias of the Liver.*

We include under these designations the hyperæmias of the liver which are developed independently of any obstruction of the circulation of the

[1] Principles and Pract. of Physic, 3d Ed., Vol. I., p. 22.

blood. They are partly of an active character, and produced by exciting causes acting upon the parenchyma of the liver; but, on the other hand, they are partly of a passive nature, being occasioned by an atony of the vessels, or by a debilitated action of the heart,[1] and in this case they are mostly of long duration. It is impossible, however, to draw a strict line of demarcation between these two forms, inasmuch as we meet with undoubted examples of a transition from the one to the other, and because we cannot always ascertain with sufficient accuracy the mechanism of the affection in individual cases. It is equally impossible to determine to what extent the capillaries of the hepatic artery are implicated along with those of the portal vein; it can scarcely be doubted, that differences exist in this respect, when we consider the varying character of the influence which hyperæmias exercise over nutrition.[2]

Congestions of the liver constitute the starting-point of almost all the structural diseases of the organ. They also induce and accompany the formation of pseudo-plasmata. An accurate knowledge, and a careful attention to these conditions are particularly important in medical practice, inasmuch as at this stage of the disease such results may be hoped for from treatment, as at a later period it is usually vain to expect.

a. Congestions proceeding from the processes of Digestion.

Even in health, the quantity of blood contained in the liver is liable to constant fluctuations, arising from the process of digestion. The increased flow of blood to the mucous membrane of the stomach and the active intestinal absorption,—both of which circumstances are necessary accompaniments of this function,—increase the pressure of blood towards the liver, and give rise to a temporary swelling of the gland, which is compensated for by the augmentation of its secretion and by an increased consumption of plasma in the cells of its parenchyma. This hyperæmia not unfrequently exceeds the boundaries of health, as when acrid, irritating matters, such as alcohol, pepper, mustard, strong coffee, &c., are taken in too large quantity. The action of spirits in this way is the one of which we know most. These liquids, if taken very frequently, give rise at last to palpable changes of structure. In our climate, they gradually lead to cirrhotic degeneration; but in hot countries, according to the experience of Annesley,[3] Twining,[4] Cambay,[5] and others, they contribute greatly to the development of suppurative hepatitis.

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[1] The simplest forms of passive accumulation of blood in the liver, which I have observed, have been in old decrepit individuals with extreme prostration of the heart's action, and in whom the abdominal muscles, from being greatly relaxed, no longer possess the property of assisting the portal circulation. It was under such circumstances that Virchow correctly pointed out that the veins of the mesentery, spleen, and stomach, are distended with dark blood. (*Virchow's Archives*, Vol. V., p. 289.) The portal system is particularly liable to suffer from such derangements, owing to its double capillary circulation, and to the comparatively scanty muscular tissue and feeble contractility of its vascular walls.

[2] Hyperæmia of the liver from abuse of spirits soon leads to cirrhotic degeneration of the gland, whilst other forms of congestion may last for a long period without producing any such results. Moreover, the circulation in the hepatic artery often appears to become deranged, and black pigment is very frequently found deposited in the finer twigs of this vessel.

[3] ANNESLEY, *Diseases of India*. London, 1828. Vol. I., p. 488.

[4] TWINING, *Diseases of Bengal*, Vol. I., p. 247.

[5] CAMBAY, *De la Dyssenterie des pays chauds*, p. 217.

The other irritating substances just mentioned, are less apt than spirits to be followed by injurious consequences, inasmuch as the ingestion of an excess of any of them is usually only a matter of occasional occurrence. Their action manifests itself chiefly by a feeling of tightness and fulness in the right hypochondrium, accompanied by a distinct enlargement of the gland, which can be made out by means of the pleximeter, and by pains which are wont to come on soon after meals, and which, as a general rule, cease after a short time. These results are chiefly observed during the warm seasons of the year, and in individuals who have already suffered from some affection of the liver, or in whom that organ is unusually irritable.

Beau [1] has, by a series of observations, endeavored to prove that severe neuralgic pains in the liver can be produced by the same exciting causes as those just mentioned,—an attempt which, in my opinion, can lead to no certain results, when we consider the difficulty of excluding the co-operation of gall-stones. We shall return hereafter to this matter in treating of Colica hepatis.

The question has been frequently discussed, in what way acrid ingesta come to act upon the parenchyma of the liver? Broussais is of opinion that the irritation of the intestinal mucous membrane is transmitted along the bile ducts to the parenchyma of the liver; others have assumed the existence of an irritation sympathetic with that of the bowel; whilst Beau first of all pointed out the direct action of irritating substances by their absorption into the portal blood.[2] It is impossible to arrive at an accurate discrimination between these different modes of action; substances which, like alcohol, are readily taken up into the blood which is about to pass through the liver, produce their effects principally by way of absorption; other substances, again, which are less easily absorbed, transmit their effects from the mucous membrane along the nerves; a direct transmission of the irritation along the bile ducts, such as was assumed by Broussais, occurs much more rarely, and is attended in most cases by other consequences, involving the excretory ducts more than the glandular substance itself, as, for instance, catarrh of the bile ducts, obstruction of the secretion, &c.

The form of hyperæmia just alluded to, usually passes off without being productive of any great mischief; it is only from its recurring repeatedly, or from its coexistence with other irritating causes, that it leads to more serious disease; even then the affection is most frequently of a chronic nature.

This last condition of chronic congestion is particularly observed in individuals who devote themselves too freely to the pleasures of the table, and who make use of a succulent stimulating diet, notwithstanding their sedentary mode of life, their great muscular inactivity, and comparatively imperfect respiration. In such cases, the absorption of nutritive material exceeds the waste, and, sooner or later, but usually in the middle period of life, the muscles become flabby, and other affections show themselves in patients with a hereditary predisposition, while even at an earlier period there arises a disproportion between the power of the heart and the quan-

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[1] Archives général. de Méd. April, 1851.

[2] Afterwards, in treating of diseases of the portal vein, we shall consider a fourth modification of the mode of transmission of pathological processes from the intestine to the liver, namely, that through the veins, which was particularly pointed out by Ribes (*Andral, op. cit.*, p. 290).

tity of blood, owing to which that part of the vascular apparatus where the greatest obstacle has to be overcome, becomes over-distended with blood. This is usually the portal system, which is the more readily congested from the fact that, under the circumstances just mentioned, the irritation of the intestinal mucous membrane, resulting from errors in diet, as also the copious intestinal absorption, contribute to the interruption of the circulation. In this way, one often sees chronic gastro-intestinal catarrh commencing with irregular, and, for the most part, retarded defæcation, and accompanied by swelling of the hæmorrhoidal veins, tympanites, and pains in the hypochondrium. To these symptoms there is frequently superadded a habitual hyperæmia of the liver, which from time to time increases and makes itself known through painful distention of the right hypochondrium, a jaundiced tinge of the conjunctiva, &c.[1] This condition may exist for a long time without inducing more serious lesions of this gland, such as fatty infiltration of the cells and catarrh of the bile ducts. In most cases, we may succeed without any difficulty in moderating or removing the hyperæmia of the liver by means of a carefully regulated diet, by avoiding all indigestible, fatty, and over-nutritious articles of diet, by increasing the waste of material by means of active exercise in the open air, riding, &c., as well as by the use of bitter laxative medicines, the solvent extracts with salines, rhubarb, aloes, and similar substances, by the application of leeches to the anus, and still better, by regulating the intestinal secretions by the use of the Springs of Kissingen, Homburg, Marienbad, and Karlsbad;[2] but if the condition is one of very old standing, it is seldom that the improvement is permanent. It would appear that the nutrition of the muscular tissue of the intestinal canal, and probably also of the lining membrane of the portal veins becomes at an early period impaired, and as a result of this, there is induced a predisposition to relapses, and the patient's complaints are protracted. Accurate examinations of the anatomical characters of the tissues just mentioned are necessary, in order to confirm and explain those circumstances which hitherto have been made the subject of theoretical speculation. Along with the mechanical derangements which have been pointed out, we usually find abnormal conditions of the metamorphosis of matter, which were formerly attributed to arthritis, to the formation of urinary gravel, &c. Our knowledge of these conditions deserves to be placed upon a firmer footing than it is at present; we shall afterwards see how far the tissue of the liver may be implicated in their development.

This is not the place to discuss in detail the various morbid processes which medical men have included under the name of abdominal plethora; when the liver is implicated, which, however, is by no means the general rule, there is in most cases nothing more than hyperæmia, or fatty degeneration (*see Chapter on Fatty Degeneration*, Vol. I., p. 215); more serious lesions, such as cirrhosis, induration, or lardaceous degeneration are rarely present. The mutual relations which exist between these affections and the condition of the intestinal mucous membrane, will be understood from what has been already stated.

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[1] Every extensive development of gas in the intestinal tube, by pressing the blood from the roots of the portal vein towards the trunk of that vessel and the liver, may induce transient hyperæmia of this organ; great accumulations of fæcal matter may give rise to similar results.

[2] See Vol. I., page 88, note.—TRANSL.

b. Traumatic Hyperæmia.

Contusion of the hepatic region is not unfrequently observed to give rise to accumulations of blood in the liver, entailing a considerable amount of swelling of the organ. Piorry has described a case of this nature resulting from the blow of a spent pistol-ball; the remarkable increase in the volume of the gland, which was accompanied by dyspnœa and fever, disappeared after a copious venesection, so soon as the following day. I have myself met with a similar enlargement of the liver in a railway laborer, the right side of whose chest had been crushed by a wagon. The recovery in this instance was more tedious; the patient continued jaundiced for three weeks, at the end of which time he was dismissed from the Hospital cured. In many cases the effects of the contusion are restricted to the part; the hyperæmia may then pass readily into inflammation, which may lead insensibly to the formation of abscess. Most of the hepatic abscesses which occur in our own climate have this traumatic origin. R. Bright has published some cases which appear to prove that the hyperæmia resulting from external violence may pass into a chronic form of inflammation, ending in induration.

c. Hyperæmias dependent upon the influence of a high temperature and miasmatic effluvia.

In hot climates, and particularly in those marshy districts which exhale deleterious substances, hyperæmias of the liver, and the injurious consequences resulting therefrom, are among the ordinary forms of disease. In addition to the high temperature, the miasmatic infection of the blood contributes very powerfully to their development; but it is a difficult matter to determine the share which is taken by each of these two agents. There are facts which would seem to show that the influence of heat has been over-estimated, owing to the idea, that in a high temperature the liver acts vicariously with the lungs. Haspel states, that in the year 1846, when the temperature in Oran was unusually high, so that the marshes were dried up and the sources of malaria destroyed, hepatic affections, in place of being more prevalent, completely ceased. In India, the prevalence of hepatic diseases depends less upon the temperature of the stations than upon their situation in the neighborhood of stagnant water and marshes. Hepatic affections usually attain their greatest frequency in autumn, when the temperature is beginning to fall. In the cold foggy climate of Holland, Pringle found conditions of the liver precisely similar to those met with in the tropics. Malaria must, therefore, be regarded as the principal exciting cause of hyperæmia of the liver, although the co-operation of a high temperature cannot be doubted.

The hyperæmia of the liver which is wont to be developed in warm climates usually attains a remarkable intensity, and, not unfrequently, in a short time induces serious structural changes. The gland is distended with dark blood; the gall ducts and bladder are filled with secretion, and in this condition hæmorrhages and sub-peritoneal effusions take place in the liver, which may end in softening of the parenchyma; in other cases, limited inflammatory deposits are formed, which, after a shorter or longer period, terminate in abscess; or derangements of nutrition arise, which give rise to hypertrophy or fatty degeneration, or, on the other hand, to

induration, or, less frequently, to cirrhotic degeneration with shrinking of the parenchyma.[1]

The hepatic hyperæmias of the tropics are often uncomplicated, but more frequently they make their appearance along with dysentery, or in conjunction with malarious fevers of an intermittent, remittent, or continuous type. In the latter case, hyperæmic swellings of the spleen, and often of the kidneys also, coexist with the hepatic affection, and in this way the various complications originate, the pathology and symptoms of which we have already briefly alluded to.

Simple hyperæmia is either acute or chronic in its progress; the derangements to which it gives rise are more marked in the former case than in the latter. Chronic hyperæmia is usually developed so slowly and insidiously that its existence is frequently not recognized until serious structural changes in the hepatic tissue, such as abscesses, induration, &c., take place, and remove all hopes of a favorable result from treatment.

The acute form of hyperæmia is characterized by more or less painful distention of the right hypochondrium, accompanied by dyspnœa, and often, also, by tension of the splenic region, and pains stretching towards the right shoulder and lumbar region. Along with these symptoms, the tongue is either clean or sometimes coated gray, and there is headache, nausea, and vomiting of mucous or green bilious matter: the bowels are irregular; in some cases they are constipated, but more frequently there is diarrhœa, with yellow, bilious, or sometimes even bloody stools: at the same time, there is great depression of spirits and rapid loss of strength, in most cases, without any increased frequency of pulse or elevation of temperature. After some days or weeks the symptoms quite disappear, or slight derangements remain, obvious only to an attentive observer, and denoting the transition of the acute into the chronic form.

Although the unpleasant sensations in the right hypochondrium may abate or disappear, the hepatic dulness still remains extensive; the appetite only partially returns, some articles of diet, particularly animal food, excite loathing, and the tendency to diarrhœa continues. These symptoms are subject to exacerbations from time to time; the hepatic tumor increases, and the gastric and intestinal derangements become more marked. Amidst alternations in the severity of these symptoms, the patients emaciate more and more, and become sad and desponding; their skin assumes a pale cachectic appearance, or sometimes there is jaundice; and dropsical effusions are poured out into the abdominal cavity, and into the subcutaneous areolar tissue. Towards the termination of the disease, there are frequently developed exhausting catarrh of the intestines, dysenteric affections, and intermittents of an irregular type, accompanied by suppuration of the parotid, &c.; or symptoms of suppurative fever, indicating the formation of abscess in the liver, may show themselves.

These symptoms of the final stage of chronic hyperæmia are mainly dependent upon the nature of the structural changes which the organ undergoes during the progress of the affection, and vary in accordance with the varying character of these changes. Upon *post-mortem* examination, the liver is sometimes found to be congested and softened, at other times it is pale and jaundiced or fatty, or it may be indurated and

[1] The deposits of pigment and their consequences, which one meets with under such circumstances, have already been treated of in Chap. VIII.

cirrhotic, or lastly, it may be penetrated by abscesses. The study of the clinical history of these pathological changes will explain the varieties in the symptoms induced by chronic hyperæmia of the liver.

In temperate climates, the affection usually runs a milder course, the symptoms are less prominently marked, and the modes of termination are different. Hyperæmia of the liver may exist here for a long period, before it gives rise to derangements of nutrition of any consequence; abscesses are scarcely ever met with. What we most frequently observe is an increase in the volume of the liver, caused by fatty deposit, or by an infiltration of the parenchyma with an albuminous substance, which gradually passes into colloid degeneration; in rare cases the liver becomes cirrhosed. Acute hyperæmia of the liver is especially observed during the hot summer months; chronic hyperæmia, with its sequelæ, is not usually developed until autumn.

The first object, in the treatment of the acute form, is to remove everything which can act by stimulating the action of the liver. The diet is to be restricted to mild demulcent or somewhat acid vegetable substances; animal food, fat, strong spices, and alcoholic drinks are to be avoided. The reduction of the excess of blood in the portal system and in the liver, is best effected by the application of leeches to the anus, tepid baths, and cooling saline purgatives, such as tamarind pulp, purified tartar, and sulphate of soda. If spontaneous diarrhœa sets in, it must not be checked too early; when it is necessary to interfere, it is best to make use of ipecacuanha, either in small divided doses, or as an emetic. The general compression to which the liver is subjected during the act of vomiting, has a great effect upon the circulation of blood through the organ. The mineral acids may also be prescribed.

When the affection is of a chronic or passive character, change of air is advisable, taking care to avoid all marshy districts. In such cases, in order to promote intestinal absorption, small doses of rhubarb, aloes, colocynth, &c., in combination with the sesquichloride of iron, and the bitter solvent extracts (Vol. I., p. 86), will be found most serviceable. An emetic may also be tried here with the object of assisting, mechanically, the circulation of the blood in the hepatic capillaries. A too strict diet is not adapted to this form of the affection: easily digested nutritious food is necessary in order to improve the condition of the blood and correct the atony of the vascular apparatus; in more advanced cases, preparations of steel are usually employed for the same purpose. Where circumstances permit, a resort to Kissingen, Homburg, or Marienbad[1] is advisable. Pringle, Lind, Portal, and Haspel have advised the application to the hepatic region of blisters, setons, and moxas, and that they should be kept open for a long period. Calomel and mercurial ointment have been frequently recommended, but must be resorted to with caution when a cachectic condition threatens to become developed. In cases where the hepatic affection is accompanied by irregular forms of intermittent fever, quinine must not be employed too hastily; the febrifuge is usually of little service until the hyperæmia of the liver has been subdued by other means.[2] When there is reason to apprehend from the symptoms, that an abscess is forming, or that lardaceous degeneration, or cirrhosis, is being developed, the treatment appropriate to each of these conditions must be adopted.

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[1] See note, Vol. I., p. 88.—TRANSL.

[2] See ANDRAL, *op. cit.*, p. 31; and also PORTAL.

Closely allied to the chronic hepatic hyperæmia of malarious regions, is the congestion of the liver which is observed in scurvy, and in similar conditions of the system, along with swelling of the spleen. Baillou[1] and Portal[2] long ago described cases of extreme distention of the hepatic vessels with dark blood, occurring in scorbutic subjects, and giving rise to softening of the glandular tissue. Andral[3] never failed to find this condition under such circumstances. According to my own observation, this morbid change in the dead bodies of scorbutic patients has not been of such constant occurrence; I have repeatedly found the organ soft, infiltrated with fat, and anæmic. Portal has recorded cases in which he obtained favorable therapeutic results from the employment of anti-scorbutic remedies combined with mild laxatives.

d. Hyperæmia of the Liver, from the Suppression of habitual Hæmorrhages.

During the climacteric period of life, the cessation of the catamenia is not unfrequently observed to be accompanied by tumefaction of the liver, which disappears with each recurrence, after a long interval, of the uterine hæmorrhage; and in this way, the swelling may return repeatedly. Like consequences are often noticed in the earlier periods of life from sudden suppression of the menses; Portal, and more recently, Henoch,[4] have published cases of this nature. The cessation of hæmorrhoidal discharges of blood, which have become habitual, may be followed by similar results. It has been ascertained, however, that this effect is by no means constant; in many cases, the liver remains quite unaffected; hence, other causes must cooperate in producing these congestions. So far as my knowledge extends, this form of hyperæmia of the liver has not been observed to give rise to any serious derangement in the nutrition of the liver; it is obvious, however, that the pre-existing diseases of the liver may be aggravated and hastened in their progress by it, and hence, in treatment, care should be taken to reproduce at an early period the original hæmorrhages, or to obtain some compensation for them.

There can be no question that there are other causes of hyperæmia of the liver, besides those already mentioned, with which we are either imperfectly, or not at all, acquainted, and that, therefore, it must very frequently happen that the cause of such an affection must remain doubtful. Hence, the forms of hyperæmia hitherto considered are not all, but merely the most important. There are three agencies in particular, which may serve for the further elucidation of these affections. First, there is the influence of the nerves, which has been in some measure experimentally proved by Claude Bernard and others, although it has not been studied by any means sufficiently, in order to be made use of in clinical investigations. That this influence may operate powerfully under certain circumstances, is apparent from the cases of diffuse inflammation and acute atrophy of the liver, which are developed immediately after mental emotions.[5] A second agency, although one that is but imperfectly understood, is the composition of the blood, the importance of which, in mala-

[1] BALLONII, *Opera*, Tom. III., p. 30.
[2] PORTAL, *op. cit.*, p. 377.
[3] ANDRAL, *Clinique Médicale*, T. II., p. 244.
[4] HENOCH, *op. cit.*, p. 85.
[5] See Vol I., page 164.

rious infection, in scurvy, &c., has already been pointed out, but which probably assists in many other cases in producing hepatic hyperæmia. Thirdly and lastly, the altered nutrition of the walls of the hepatic vessels, which hitherto has attracted but little attention, promises to explain many points, not only in the chronic forms of hyperæmia of the liver, which are for the most part dependent upon atony of the blood-vessels, but also in the partial congestions and apoplexies of the organ. These causes of hyperæmia must be reserved for further investigation.

As an appendix to the hyperæmias, we shall consider as briefly as possible :—

Hæmorrhage from the Liver.—Apoplexy and Hæmorrhagic Softening of the Liver.

These affections are in general of rare occurrence, and are for the most part the result of intense congestions, which are induced by miasmatic infection of the blood. Hence they are particularly observed as a complication of the malarious fevers of the tropics, and occasionally, also, of the malignant intermittent fevers of colder climates. The extravasated blood is sometimes accumulated in isolated masses, or circumscribed beneath the serous covering of the gland, whereas at other times the parenchyma is more uniformly infiltrated with blood, and converted into a dark, abnormally soft, and, at some places, pultaceous mass, in which the remains of the normal tissue can only be sparingly recognized. The gall-bladder usually contains a dark, thick secretion.[1] This condition is accompanied during life by the symptoms of malignant marsh fever, by pains in the right hypochondrium, and almost always by jaundice and bilious vomiting; the coexistence of hæmorrhages from other parts of the body is not uncommon. Annesley, Haspel, and others, record many examples of this nature.

Similar morbid changes in the liver have occasionally been observed in scurvy and allied affections. Extravasations of blood in the hepatic tissue, or beneath its capsule, are also frequently caused by external violence, and occasionally by obstructive hyperæmia; they are particularly observed in new-born children after tedious deliveries, and in conjunction with atelectasis of the lungs.[2]

There are some very remarkable cases of hæmorrhage of the liver, where, without any pre-existing complaints, the bleedings have come on suddenly, and in a short space of time have terminated in death. Andral[3] records instances of this nature which do not admit of any certain explanation. One case was that of the Superintendent of the Mint at Paris, who, on awaking in the morning, complained of slight uneasiness, and expressed a desire to remain in bed. Some hours after he was found

[1] It has not yet been determined with sufficient accuracy to what extent the extravasated blood enters the hepatic ducts and is conveyed by them to the gall-bladder and intestine, or whether it does so at all. Saunders believed that he had found blood in the bile in several cases; this secretion was almost black, coagulated, and deposited, upon the addition of water, a sediment consisting of red corpuscles.

[2] In a stillborn child, with hernia umbilicalis congenita, I have found upon the convex surface of the left lobe of the liver a sub-peritoneal extravasation of blood, 1¼ inch in diameter, and at the same time smaller extravasations in the substance of the gland. See further, F. Weber, *Beiträge zur patholog. Anat. der Neugeborenen*, Bd. III., S. 56.

[3] ANDRAL, Clinique Médicale, T. II., p. 247.

dead. On *post-mortem* examination, the organs of the head and chest appeared perfectly normal; the abdominal cavity contained a large quantity of partially coagulated blood. In the middle of the convex surface of the right lobe of the liver an opening was visible the size of the point of the finger; this led into a cavity the size of a hen's egg, filled with blood, at the bottom of which was detected the ruptured wall of a large branch of the portal vein; in other respects the liver was healthy. Andral unfortunately makes no mention of the condition of the coats of the vena porta. In another case, described by Honoré, the liver contained several cavities filled with blood; no search for a ruptured vessel was made. Louis[1] found in the liver a cavity the size of a nut, filled with coagula of blood arranged in concentric laminæ. It is probable, that these hæmorrhages depend upon local diseases of the vascular coats, which have not yet been sufficiently examined. In the Third Volume of this work, I shall communicate a case of fatty degeneration of the coats of the portal vein, which occasioned rupture of this vessel before its entrance into the liver, and, as a consequence of this, fatal hæmorrhage. The coats of this vein may be subject to similar derangements of nutrition in the interior of the liver.

One additional case of hæmorrhage from the liver may be here mentioned in detail.

Observation No. XVII.

Tedious delivery.—Symptoms of Peritonitis.—Jaundice.—Vomiting of black flakes.—Delirium.—Death.

Purulent effusion in the peritoneal cavity.—Hæmorrhagic softening of the Liver.—Extravasation of blood beneath the capsule.

Caroline Herbst, aged 38, on January 10th, 1856, was confined for the twelfth time; labor was tedious. The very day after, vomiting and diarrhœa, with suppression of the lochial discharge, made their appearance.

On the patient's admission into Hospital on the 18th, the abdomen was found remarkably distended and tympanitic; dulness upon percussion could be made out in the iliac regions; there was a slight amount of pain; the uterus extended a hand's-breadth above the symphysis of the pubes, the liver and spleen were of normal dimensions; the stools were brown, hard, and scybalous. The organs of the chest were normal; pulse 110 and small. Was ordered tamarinds with sulphate of soda and warm cataplasms.

On the 19th, the bloody discharge from the genital organs returned; meanwhile, the right hypochondrium had become painful; the pulse had risen to 120, and there was slight diminution of the tympanites; the conjunctiva and the countenance had assumed a jaundiced hue. The bowels had not been moved. Continuation of the same remedies, and an enema.

On the 20th, the jaundice was very intense, and was accompanied by

[1] Louis, Recherch. anatomico-patholog., 1826, p. 381.

typhoid somnolence, great prostration, and repeated vomiting of brown flaky masses. The urine drawn off by catheter abounded in bile-pigment, was acid, and free from albumen, and had a specific gravity of 1014. Was ordered phosphoric acid and analeptics. The tympanites and somnolence increased, and death ensued on the following morning.

Autopsy, 17 hours after death.

The body was of a dark-yellow color; there were a few patches of putrefaction. There were no important morbid changes in the organs of the head or thorax; the heart contained dark, firmly coagulated blood. A considerable quantity of purulent fluid distinctly tinged with bile was effused into the peritoneal sac; the peritoneum was opaque and injected.

A grayish-yellow fluid, mingled with black flakes, was found in the stomach; the mucous membrane was pale, and free from ulceration; the intestinal canal contained at its upper part, thin and yellow, and at its lower, firm and brown, fæcal matter; the mucous membrane was normal.

The spleen was unaltered in size and consistence; it weighed 0.17 kilogr. (6 oz. avoird.). The kidneys were of a jaundiced tint, but in other respects normal.

The uterus extended about three inches above the symphysis pubis, and its inner surface was covered with a reddish-brown fluid, but presented no morbid change of any consequence; the veins and lymphatics were empty; the left ovary contained a cyst the size of a walnut, filled with a clear fluid; the vagina was of a livid hue, without any exudation.

The liver appeared enlarged, it weighed 2.3 kilogr. (5 lbs. 1¼ oz. avoird.); its form was not much altered; its consistence was soft and flabby, and some parts of the right lobe and lobulus quadratus felt pultaceous. Beneath the serous covering, numerous extravasations of blood were observed, varying in size from that of a groschen (6¼ Eng. lines, or rather less than a sixpence), to that of a thaler (1¼ inch, somewhat larger than a half-crown); a flabby, bladder-like elevation of the capsule, filled with bloody fluid, and more than an inch and a-half in diameter, was situated at the outer margin of the right lobe. Numerous large and small extravasations of blood were found in the parenchyma of the gland; the glandular tissue was at some places reddish-yellow or yellow, and exhibited distinctly the outlines of the lobules; at other places, it was of a dirty reddish-brown color. A large quantity of bloody serous fluid exuded from the cut surface, especially from the deeply injected portal margins of the lobules. The hepatic cells in the centre of the lobules were normal; at the periphery they were mingled with a quantity of debris and free nuclei; many of them contained oil globules; there was very little bile-pigment, either in a granular, or in a diffused form.

The bile ducts contained a little pale-yellow mucus; and the gall-bladder, a small quantity of thick, greenish-yellow bile, free from albumen. The branches of the portal vein, which were traced as far as was practicable, were of normal character, and the fluid blood contained in them presented nothing unusual under the microscope.

This case, so far as regards the lesion of the hepatic tissue, was allied to the changes which the liver is wont to undergo in the fevers of tropical climates and in scurvy. No cause, however, of this nature could be made out, while the idea of a putrid infection of the blood was countenanced by

neither the symptoms nor the anatomical lesions. The intense hyperæmia of the liver could not be attributed to the suppression of the lochia, because the jaundice did not make its appearance until this discharge had returned. It would seem that the same condition was present in this case, as, in a milder form, not unfrequently induces diffuse hepatitis and acute atrophy of the liver in pregnant females. The incipient disintegration of the hepatic cells at the margin of the lobules, the deposit of fat in these cells, and the infiltration with fluid exudation, are circumstances which render this view of the case more probable than any other.

CHAPTER III.

INFLAMMATION OF THE LIVER. ITS VARIOUS FORMS AND CONSEQUENCES.

HISTORICAL ACCOUNT.

By the term inflammation of the liver, the ancient physicians designated certain groups of functional derangements, with the anatomical origin of which they were but imperfectly acquainted.[1] Hence an indefinite idea was attached to the term, which comprehended many diseases that did not properly belong to it. Of the earlier observations, those only can with certainty be relied on which proved to be really instances of inflammation, by terminating in the formation of abscess; cases of this nature were long ago described by Hippocrates, and his description was accompanied by some very apposite observations on diagnosis and prognosis. Galen (*De locis affectis*, Lib. V. Cap. 7) distinguished between phlegmon and erysipelas of the liver, and, in addition to inflammation, described a cold and a hot "intemperies." Bianchi designated this intemperies by the term hepatitis,[2] and made phlegmon and erysipelas of the liver distinct from it.[3] In this way, writers fell into the error of making artificial subdivisions, for which no real foundation existed in nature. It was not until the seventeenth century, when pathological anatomy began to be studied, that a firm foundation was afforded for the clinical observation of these affections; but still, for a long period, physicians applied the term hepatitis to a group of symptoms, which in many instances did not arise from inflammation of the liver;[4] and, even at the present day, practitioners employ the term inflammation of the liver far more frequently, than is warranted by the circumstances of the case.

By means of anatomical investigations, the materials have been gradually collected, from which our present knowledge of hepatic inflammation is derived. The more obvious lesions were first determined. Dodonæus, Bartholin, Ballonius, Guy-Patin, Bonet, Manget, Valsalva, and others, recorded observations of hepatic abscess, so that Morgagni (*Epistola* 36) was enabled to bring together a long series of them, and to draw valuable conclusions from them, as to the various modes in which the abscesses

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[1] GALEN (*Definit. med.* No. 274): Hepatici sunt quos jecoris dolor comitatur diuturnus cum tumore et duritie et corporis decoloratione; supervenit illis febris ardens et lingua exarescit.

[2] BIANCHI, *loc. cit.*, p. 149: Hepatitis est inflammatio hepatis non exquisite legitima. He mentions three varieties: Hepatitis calida, frigida, et mixta.

[3] *l. c.*, p. 338: Erysipelas hepatis est inflammatio latior et acrior et totum occupans viscus, neque in peculiarem tumorem coacervata.

[4] VAN SWIETEN (*Comment.*, Tom. III. p. 81): Hodie plerique medici acutos hepatis morbos sub hepatitidis nomine comprehendere solent. Boerhaave and Van Swieten discussed the entire pathology of the liver under the title "Hepatitis et Icterus multiplex."

open, and as to their concomitant symptoms. Besides the formation of abscess, induration was recognized at an early period, as an effect of hepatitis; this, however, was not distinguished from scirrhus and true cancer. Even Portal (*Maladies du Foie.* Paris, 1813, p. 267) enumerates as the consequences of hepatitis, suppuration, induration, scirrhus, ulceration, cancer, and gangrene. In the course of time, medical men learnt to distinguish the inflammatory affections of the bile-ducts, of the capsule of the liver, and lastly, of the hepatic vessels from those of the glandular parenchyma, and thus, the region of hepatitis proper was gradually more and more circumscribed. As a consequence of this, observers were forced to the conclusion, that in practice the diagnosis of inflammation of the liver was made far too frequently, and that many of the symptoms, which had been thought to indicate its presence, were only due to a hyperæmia of the gland, a catarrh of the bile-ducts, or to an inflammatory condition of the serous covering, or of the hepatic vessels.[1] It is true, that in more recent times, Bonet (*Traité des maladies du Foie.* Paris, 1841) endeavored to show that almost all the forms of diseases of the liver arise from irritation and inflammation of that organ; but, inasmuch as his hypothesis was obscure, and not based upon observation of facts, it met with little support. In our own country, but few observations could be made on true inflammation of the liver terminating in suppuration, owing to the rarity with which the lesion is met with: the works of Abercrombie (*op. cit.*), Louis (*Mémoires ou Recherches Anatomico-Pathol. sur Diverses Maladies*), Andral (*Cliniq. Médic.* Tom. II.), and others, contain a comparatively small number of observations of such cases. This form of inflammation, indeed, is only prevalent in tropical countries, and we have to thank the physicians who have practiced there for the best works on suppurative hepatitis. Such are the works of Annesley (*Researches into the Causes, Nature, and Treatment of the more prevalent Diseases of India.* London, 1841); Cambay (*Traité de la Dyssenterie des Pays Chauds.* Paris, 1847); Haspel (*Maladies de l'Algérie.* Paris, 1852. Tom. II.); Charles Morehead (*Clinical Researches on Diseases in India.* Vol. II. London, 1856); and of some of the earlier authors, as Bontius (*De medicina Indorum.* Pars III. Cap. VII. 1645), W. Saunders (*Observations on hepatitis in India.* London, 1809), Griffith, and others.

In our own climate, as likewise in all countries in the Temperate Zone, there are two other forms of inflammation of the liver, of far more importance than suppurative hepatitis; of these, one terminates in simple or granular induration, the other in softening and acute atrophy of the gland. The former of these was known to the ancients, but has only in recent times been made the subject of close investigation; it has a clinical history of its own. The second, however, has only been recognized as a form of inflammation, and closely studied, in our own day; its history coincides, for the most part, with that of malignant typhoid jaundice, which has already been given in the chapter on Acholia in the first volume. (See Vol. I., Chap. V., p. 136.)

DIFFERENT FORMS OF INFLAMMATION OF THE LIVER.

Inflammation of the liver is a protean malady, difficult to treat of, owing to the fact that the individual parts only of the complex organ may be

[1] Fr. Hoffmann (*Opera Omnia Physico-medica*, Tom. V.) long ago expressed a similar opinion in these words:—"Hepatis phlegmonem si non in entium, tamen rarissimorum affectuum classem referendam esse."

diseased, and because, in addition to this, the process itself may vary greatly in its intensity, extent, and consequences. The inflammation may have its seat in the fibrous envelope of the gland, or in the sheath of the vessels in Glisson's capsule; or it may attack the glandular parenchyma, the vessels (the portal or hepatic veins), or lastly, the bile-ducts.

Of the last two of these forms we shall say nothing at present, but shall reserve them for subsequent consideration.

Inflammation of the hepatic parenchyma is either circumscribed, leading to abscess or to granular contraction, or it is diffusely extended over the entire organ, and then, according as the process uniformly involves all the anatomical elements of the gland, or is limited to the areolar matrix between the lobules, it gives rise, sometimes to softening and acute atrophy, or at other times to induration or cirrhotic degeneration.

I. INFLAMMATION OF THE CAPSULE OF THE LIVER AND OF GLISSON'S CAPSULE (PERI-HEPATITIS, PERITONITIS HEPATICA.)

1. *Its nature.*

We frequently find the remains of inflammatory processes in the capsule of the liver and its prolongations; they are rarely, however, accompanied by serious derangements, and it is only in exceptional cases that they lead to dangerous results. This only happens,[1] so far as my experience extends, when the inflammation attacks the portal or hepatic veins, or causes obstruction of the large bile ducts—events which, on the whole, are of very rare occurrence.

2. *Causes and consequences.*

Peri-hepatitis may proceed from various causes. In the first place, one sees it as a part of general peritonitis, in which case it is followed by no important consequences. The capsule is found covered with a gray exudation, or with a layer of pus, or, in the case of tubercular and cancerous peritonitis, with little nodules, which do not in any essential degree impair the function of the organ; in a few cases only have I observed circumscribed collections of pus lying upon the convex surface of the gland, which had induced a slight atrophy of the parenchyma; in one instance, a cancerous peritonitis had penetrated deeply into the substance of the liver, without, however, causing any obstruction to the circulation of the blood or to the excretion of bile. In rare cases, the inflammation is the result of external violence directed against the hepatic region; it then gives rise to circumscribed thickenings of the capsule, usually only involving the superficial layer of the gland, but sometimes leading to a deep furrow on the surface of the organ.

More frequently, disease of the liver itself is the cause of the inflammation; in most cases of abscess of the liver, and of simple or cirrhotic induration, the capsule is found thickened, and united by numerous bands of areolar tissue to the neighboring peritoneum, the surfaces of the ribs,

[1] Andral (*Clinique Méd.*, Tom. IV. p. 310) relates a case where the formation of pus upon the capsule of the liver, from its spreading to the peritoneum, gave rise to fatal peritonitis.

or to the adjacent portions of the bowel. This result is of far less frequent occurrence in cancer and in echinococci of the liver; these diseases often attain a remarkable extent, without producing any adhesion or thickening of the capsule.

In many cases, peri-hepatitis is an inflammatory process which has spread from some of the neighboring organs; for example, in cases of right pleurisy, we occasionally observe a participation of the serous covering of the diaphragm and of the liver, and in simple and cancerous ulceration of the stomach the inflammation may be observed to extend to the capsule of the liver along the hepato-duodenal ligament, or from the small curvature of the stomach along the coronary ligament, in the former case extending with Glisson's capsule deep into the substance of the liver, and in the latter, occasionally implicating the vena cava and hepatic vein.

Thickenings of Glisson's capsule often occur without any very obvious cause. The portal vein and hepatic artery, as well as the nerves, are

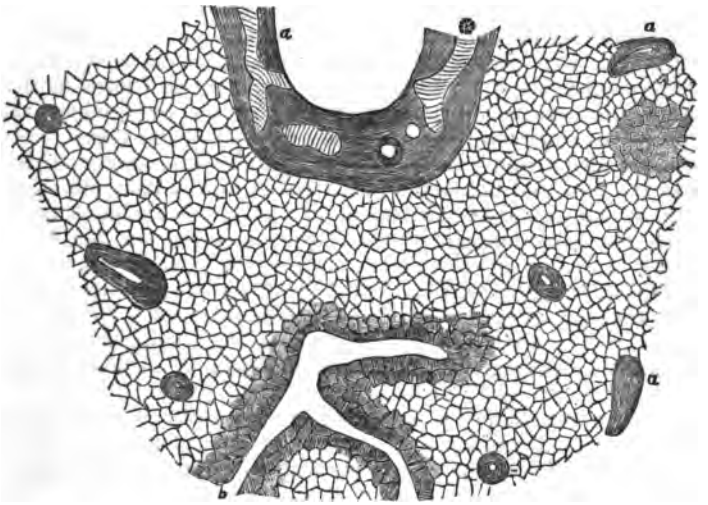

FIG. 8.—A magnified thin section of a liver, showing the changes in its structure produced by chronic atrophy. The sheaths of the branches of the portal vein (a) are seen to be remarkably thickened, and form a striking contrast to the thin walls of the hepatic vein (b). At *, hepatic arteries are represented as contained in the sheath of a branch of the portal vein.

found enveloped in a firm sheath,[1] which extends even to their finest ramifications, in most cases without altering in any way the calibre of these vessels or the parenchyma of the gland. I have never, in these cases, observed a distinctly granular appearance of the parenchyma, and I cannot, therefore, subscribe to the view of those who refer cirrhosis of the liver to thickening of Glisson's capsule.

[1] In one case, I have observed a dry, cheese-like pus in Glisson's capsule. In another case, where death had been preceded by general fever, loss of appetite, short, dry cough and typhoid symptoms, but where no local lesion could be made out during life, Cruveilhier found the areolar tissue surrounding the portal vein infiltrated with pus, and small abscesses along the course of the vessels of the meso-colon and meso-rectum. Here, the inflammation of the sheath of the vessels, had extended from parts at a distance to the liver, the glandular substance of which, however, still remained intact.

On the other hand, the process in many cases acts injuriously upon the portal vein. I have repeatedly found the trunk and branches of this vessel remarkably thickened, whilst the implication of the small twigs has been manifest from their rough, brown-colored lining membrane. The capillary vascular system has been destroyed over extensive tracts, and the parenchyma atrophied and shrunk. (See Chapter on Chronic Atrophy, Vol. I., p. 177, Observation No. XXIII.)

In one case, this condition of the vena cava was much more marked and the branches of the hepatic vein were likewise implicated; here the inflammation had spread from the lining membrane of the diaphragm, and had ultimately produced obliteration of the smallest branches of the hepatic veins. The lining membrane of the vena cava I found corrugated in folds, and the folds at some places adherent to one another by bands of areolar tissue; the sheath of the hepatic veins was considerably thickened, and in their interior was observed a firmly-adherent coagulum, which filled up the greater portion of the calibre of the vessel; several branches of this vessel were completely obliterated. This case, which shall be described in detail in the chapter on the Diseases of the Vessels of the Liver, terminated fatally under symptoms of obliteration of the portal vein. The posterior margin of the liver was intimately adherent to the diaphragm by thick, firm layers of areolar tissue. Under similar circumstances, I have noticed inflammatory processes on the concave surface of the liver, which, through the cicatrix-like contraction of the newly-formed areolar tissue, h· d sometimes caused dislocation (*Zerrung*) of the gall-bladder, impeded the evacuation of the bile, and led to the formation of biliary concretions, and, at other times, had produced obliteration of the ductus choledochus, and so given rise to a fatal stoppage of bile. (See Observation No. VII., Vol. I., p. 102.) In one case of a similar nature, the portal vein was obliterated. (Observation No. XXX., Vol. I., p. 191.) It is worthy of notice, however, that notwithstanding considerable thickening of these parts, the calibre of the bile-ducts and of the vessels frequently remains unaltered, and that they are often found enlarged, in place of being contracted.

Although peri-hepatitis is in general a very trifling affection, it may, under certain circumstances, in the manner just explained, become a dangerous disease. Hence the symptoms which indicate its existence ought never to be neglected.

3. *Symptoms.*

The chief symptoms, by which the disease may be recognized, are the following. First, there is tenderness of the hepatic region upon pressure, upon motion, and upon deep inspiration, without any change in the volume or situation of the organ.[1] Jaundice, as a rule, is absent, or is slight and of short duration. In addition, there are the symptoms of the primary disease, simple ulcer or cancer of the stomach, right pleurisy, &c. Febrile excitement of the vascular system is not unfrequently present. When the portal vein, the hepatic veins, or the bile ducts become implicated, the symptoms of disease of the vessels of the liver, or of chronic atrophy, or of obstruction of bile manifest themselves.

[1] In the case of collections of pus lying circumscribed between the diaphragm and the liver, the gland is pressed downwards in a similar manner to what it is in empyema on the right side.

4. *Treatment.*

As to treatment, local abstraction of blood, warm cataplasms, calomel, the neutral salts, a rigid diet, and rest, together with a due regard to the indications for treatment derived from the primary disease, are the best means for combating this form of hepatic inflammation. The injurious consequences to which the inflammation may lead, require a treatment corresponding to their nature, which, in most cases, however, must be quite ineffectual.

II. INFLAMMATION OF THE HEPATIC PARENCHYMA.

This occurs either as a circumscribed process, limited to isolated patches; or it is diffuse, extending over the entire organ in a more or less uniform manner. The former variety leads in most cases to suppuration and the formation of abscess; the latter, on the other hand, at one time induces rapid destruction of the glandular elements with softening and atrophy of the organ, and, at another, induration and cirrhotic degeneration.

A. DIFFUSE INFLAMMATION OF THE LIVER.

a. The Acute Form.

(*Hepatitis Diffusa Parenchymatosa.*)

In explaining the origin of acute atrophy of the liver, we have already shown that the destruction of the hepatic cells, and the rapid diminution in the size of the gland, are produced by an exudation process extending over the entire organ. It may be difficult to furnish any proof of the existence of the exudation, when the process has passed through all its stages, and the gland is already atrophied; it can only be detected when the disease is examined in its primary stage. In addition to patches of hyperæmia, we then find the peripheral portions of the lobules infiltrated with gray matter, whilst a fluid rich in albumen flows from the cut surface of the gland, and the capsule appears rough and opaque.[1]

This infiltration of the hepatic parenchyma with albuminous matter by no means always leads to rapid diminution in volume, or to acute atrophy of the gland; in two cases, I have found the organ enlarged (Observations I. and II., Vol. I.); in another, its size was scarcely altered, but it was very much softened (Observation III., Vol. I.); a destruction of the glandular cells, however, could be made out in all cases. Whether atrophy of the gland occurs or not, depends partly on the stage at which the disease becomes fatal, and partly upon the condition of the liver at the commencement of the process of infiltration; previous fatty degeneration, or hypertrophy of the areolar framework, modifies essentially the anatomical results of the albuminous exudation, as is shown by Cases I. and II.

This morbid state of the liver is usually accompanied by a similar con-

[1] See Vol. I., p. 157, for the detailed description.

dition of the kidneys and spleen. The epithelium-cells of the former organs undergo fatty degeneration and become small, and, in some cases, large quantities of albumen pass off in the urine: the spleen is found in a state of acute tumefaction.

This morbid state is observed, in the first place, after violent mental emotions, where the disordered innervation appears to induce the disease; in the next place, it is particularly frequent in pregnant females; and, lastly, it occurs in blood-poisonings resulting from typhus, pyæmia,[1] and allied processes. Graves (*Clinical Medicine*, p. 569) and Budd (*Diseases of the Liver*, 3rd edition, p. 169) have observed symptoms indicative of a diffuse inflammation of the liver — tumefaction and tenderness of the gland, jaundice, &c.—soon after the appearance of the eruption of scarlet fever. The cases, however, terminated favorably, and no opportunity was afforded of ascertaining the real nature of the hepatic lesion by anatomical observation.

Whether, and to what extent, the softenings of the liver, which have been observed by Annesley, Haspel, and others, as concomitants of tropical malarious fevers terminating unfavorably, are of this nature, cannot be determined, until we are furnished with more accurate investigations of the *post-mortem* appearances. (See Obs. No. XX.)

The destruction of the liver, which takes place under such circumstances, is indicated during life, not only by the symptoms of atrophy already described, but also by those peculiar changes in the urine, which, at the height of the disease, are sufficient of themselves for enabling us to form a correct diagnosis. These changes are present, even when no real atrophy of the organ results from the diffuse inflammation.

I give here the details of two cases of diffuse inflammation; one of which is also very interesting from the fact, that, in addition to the general inflammation of the glandular tissue, isolated, circumscribed masses were present, constituting a transition into *hepatitis circumscripta*.

Observation No. XVIII.

Dipsomania and irregular habits of life.—Persistent derangements of digestion.—Jaundice.—Enlarged Liver.—Somnolence.—Noisy delirium.—Coma.—Death.

Autopsy.—Enlarged Liver, with circumscribed masses of inflamed tissue scattered through it.—Destruction of the cells, and hypertrophy of the areolar framework.—Small Spleen.—Extravasations of Blood in the Lungs, beneath the Pleura and the Epicardium.—Fatty degeneration of the muscular tissue of the Heart and of the Kidneys.—Urine abounding in Tyrosine, Kreatine, and Leucine, and emitting an odor of sulphuretted hydrogen.

C. Solinsky, a mason, aged 36, who had been a great drinker, was brought, in an unconscious state, into All Saints' Hospital, on October 13th, 1858. His wife stated, that several years before, he had undergone a severe attack of cholera, and that latterly he had often suffered from pains in the stomach and in the loins, and likewise from vomiting and

[1] In most cases of pyæmia, it is only the first stage of the process,—the granular infiltration of the hepatic cells,—which can be made out.

diarrhœa. His habits had been extremely irregular. On the 8th, he had been seen in the street, apparently well. What he had been doing from the 8th to the 13th of October, no one knew.

The patient presented a slight jaundiced tint and a pasty aspect; over the hepatic region there lay a large turpentine plaister. He was very drowsy, answered questions, either not at all, or irrationally, and repeated the same senseless expressions several times; there was no paralysis. The pupils were of normal size and reaction under the influence of light. Pulse 78 and small; heart sounds free from abnormal bruit; respirations 24. The liver was somewhat enlarged, its dulness in the mammary line amounting to 16 centimètres (6¼ English inches), and in the sternal line to 12 (4¾ English inches); no tumefaction of the spleen; epigastrium greatly distended by gas. The bladder contained a large quantity of brown urine, which smelt of sulphuretted hydrogen, had a feebly acid reaction, and contained a small quantity of bile-pigment, but no albumen. Bowels confined.

The patient was ordered to take Muriatic Acid, and also one ounce of Castor Oil.

In the course of the day, the jaundice increased, and the patient became restless and loudly delirious, beat about him, and was with difficulty retained in bed.

In the night, he became quieter, and ultimately fell into a deep coma. Was ordered Decoction of Colocynth, to alternate with the Muriatic Acid. On the 14th, pulse 96, and respirations 24. The patient could not be roused; skin cool and of a sulphur-yellow color; no alteration in the size of the liver and spleen; no evacuation of the bowels; the urine drawn off by catheter, smelt strongly of sulphuretted hydrogen, colored the silver instrument black, had an acid reaction, abounded in the coloring-matter of bile, was free from albumen, and had a specific gravity of 1020.

Towards noon, death occurred under symptoms of cerebral paralysis.

Autopsy, 16 *hours after death.*

The cerebral membranes and substances of the brain presented a normal appearance; the left internal carotid artery was atheromatous. At the base of the skull there was an ounce and a-half of clear fluid of a jaundiced tint.

The bronchi were slightly injected, and covered with a white frothy mucus. The pleural cavities contained several ounces of bloody, serous effusion. Both lungs were congested, and scattered through their tissue were extravasations of blood, varying in size from a pea to a walnut. Several ecchymoses were also observed on the pericardium. There was a quantity of firmly coagulated blood in the right side of the heart; the valves on both sides were normal; the muscular tissue was flabby, friable, and fatty; there were small ecchymoses beneath the epicardium.

The mucous membrane of the stomach was of a dirty-gray tint; in the neighborhood of the pylorus it was livid. The lining membrane of the small and large intestines was pale, and the colon contained a large quantity of clay-like fæces.

The spleen was small and flabby, 4½ inches long, and 2¼ inches broad, and 1 inch thick; its capsule was wrinkled; its parenchyma soft and reddish brown.

Pancreas normal.

The kidneys were anæmic and somewhat enlarged; their cortical substance was of a grayish-yellow color, and their epithelium was loaded with fat. The urinary bladder contained a large quantity of brown, acid urine, free from albumen, and no longer smelling of sulphuretted hydrogen.

The liver weighed 2.1 kilogrammes (4 pounds 10 ounces avoirdupois), and measured 13 (Paris[1]) inches in its transverse diameter, the right and the left lobe each measuring 6½ inches; from behind forwards, the right lobe measured 6¾ inches, and the left 4½ inches; the thickness of the right lobe amounted to 3 inches. The margins were sharp, and the surface was covered with flat projections, of the size of a five-groschen piece (or about the size of a shilling), some of them smaller; these prominences had an ochre-yellow color, and faint pale-yellow edges. On the convexity of the right lobe one triangular mass was found presenting a blood-red color, measuring 1 inch by 1½ inch, and penetrating 1½ inch into the substance of the hepatic tissue; this mass contained in its centre a vessel filled with coagulated blood, and was surrounded by a yellow rim. The lobules of the yellow-colored masses, and likewise those in the rim of the red mass, were larger than those in other parts, and were separated from one another by gray rims infiltrated with serum. Similar rims, exuding a serous fluid upon pressure, could be distinguished throughout the entire gland, the cut surface of which exhibited in consequence a peculiar appearance. The adipose cellular tissue of the gall-bladder, which contained about half-an-ounce of greenish-brown fluid, was likewise œdematous. The consistence of the liver was doughy and tenacious. On closer examination, the secreting cells were found to be everywhere destroyed, and their place supplied by numerous oil-globules, granules, and particles of coloring matter. The areolar framework, in which the glandular cells were imbedded, was considerably thickened, so that large meshes remained after removal of the débris of the cells by means of boiling ether. (Fig. 2.)

The tenacious consistence of the organ was thus accounted for. Nothing abnormal could be discovered in the hepatic artery, or in the portal vein; the latter vessel appeared very anæmic.

The urine, which had been secreted a few hours before death, was subjected to further examination. On drying a few drops upon an object-glass, numerous sheaf-like crystals of tyrosine separated, and along with these, prismatic crystals, which were afterwards ascertained to be those of kreatine. Plate I., Figs. 4 and 5.

Paper saturated with acetate of lead was colored black by the vapor from the boiling urine. When evaporated to one-sixth of its volume, the urine deposited a large quantity of tyrosine, 1.5 gramme (23 grains troy) being obtained from 250 cubic centimètres (nearly 9 fluid ounces.) Along with this, there were numerous crystals of kreatine and oxalate of lime. It was remarkable that, upon further condensation, no distinct separation of leucine took place. Upon the addition of alcohol, the urine deposited a large quantity of a white flaky substance, similar to that observed in the case of acute atrophy of the liver. (See Observation No. XV., Vol. I., p. 142.) This substance was amorphous; no cystine nor taurine could be detected in it; when dissolved in water and evaporated for a long time, crystals of kreatine and oxalate of lime appeared, together with a large quantity of amorphous material. When the urine, mixed with alcohol,

[1] See Vol. I., p. 13, *Note*.—TRANSL.

was allowed to stand for a long time, the greater portion of the flaky substance was again dissolved; this fluid was reduced to the consistence of syrup, and set aside to crystallize. An abundant deposit of leucine first took place, whilst the amorphous matter, precipitable by alcohol, had disappeared, leaving scarcely a trace behind. Only small traces of urea could be detected in the urine.

Kreatine, leucine, and tyrosine, were found in the blood of the heart of the axillary vessels, but no urea; these three substances were also found in the parenchyma of the kidneys, but no kreatine could be detected in the fluid of the pericardium.

This case is interesting in several respects. The changes in the parenchyma of the liver were partly of old, and partly of recent date. The hypertrophy of the areolar matrix, and also the large quantity of fat contained in the organ, must be regarded as the consequence of the patient's intemperate habits, whilst the serous infiltration, and the destruction of

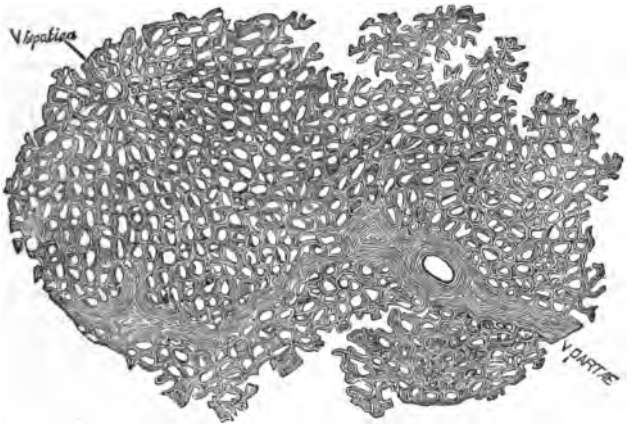

FIG. 4.—Thin section of the liver described in Obs. XVIII. The débris of the glandular cells has been removed by means of boiling ether, leaving behind the fibrous matrix, which is greatly hypertrophied.

the hepatic cells were of an acute nature. Softening or disintegration of the gland did not result from the structural changes, existing previous to the supervention of the acute process.

The large quantity of kreatine contained in the urine is an important fact, which we have already succeeded in confirming in several cases, where the urea of the urine had greatly diminished or entirely disappeared. Ludwig and Hermann[1] have made experiments by tying the ureters of animals, which throw a new light upon this matter. After applying a ligature to the ureter, they found little urea and a large quantity kreatine; whilst the latter disappeared, and the urea increased, after the ureter had been again free for some hours. In one case, where the ligature had been allowed to remain 96 hours, neither urea nor kreatine could be detected, but only a substance resembling leucine.

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[1] Sitzungsbericht der mathematisch-naturwissenschaftlichen Classe der kaiserlichen Akademie in Wien, Bd. XXXVI. s. 349.

Observation No. XIX.

Pains in the Epigastrium.— Vomiting.—Slight Fever.—Enlarged Liver. —No tumefaction of Spleen.—Jaundice.—Petechiæ.—Hæmatemesis. —Somnolence.—Death.

Autopsy.—Large, Fatty, and Jaundiced Liver, with disintegrating cells and pervious bile-ducts.—Ecchymoses beneath the pleura and epicardium.—Small Spleen.—Fatty Kidneys.

Emil. Gröbler, aged 21, a bookkeeper, a strongly-built muscular man, three months before his present attack had suffered from jaundice, which lasted for several weeks, and was accompanied by dyspeptic symptoms; since then, the young man had been perfectly well. On the 2d of October, 1858, he was seized with pains at the scrobiculus cordis, nausea, and slight febrile symptoms, and, during the following night, with repeated vomiting. At the time of his admission into the Hospital on the 3rd, the vomiting had ceased; the epigastrium was still tender upon pressure, and tense; the liver was enlarged; its margin could be easily traced with the finger far into the right hypochondrium; no enlargement of the spleen could be made out. The temperature of the skin was but slightly elevated. Pulse 96; moderate headache; consciousness unimpaired. Was ordered saturation [1] and warm cataplasms to the region of the stomach.

On the 4th, the conjunctiva, and the skin of the face and chest, were observed to be slightly jaundiced. The dark-red urine, however, was free from bile-pigment and albumen; the stools were firm, and of a dark-yellow color.

On the 5th, the jaundice became more distinct, and the urine presented the reaction of the coloring-matter of bile. The epigastrium was no longer tender; but the liver was still enlarged and easily felt, whilst the spleen was small and compressed backwards. Numerous petechiæ were observed on the skin of the chest and extremities. In the afternoon vomiting came on, and about a pound of blood, presenting the appearance of coffee-grounds, was thrown up. The patient exhibited great indifference to his condition, but his consciousness was unimpared. Was ordered dilute Hydrochloric Acid in Decoction of Althæa.

On the 6th, the pulse was 110, and there was slight elevation of the temperature; the vomiting had ceased; the patient began to be drowsy, and supposed he was getting better. Towards noon, the pulse became more frequent, and smaller; complete stupor set in, which, about two o'clock, terminated in death.

Autopsy, 20 hours after death.

A moderate degree of jaundice of the skin and conjunctiva.

Dura mater yellowish; pia mater injected; the substance of the brain was of somewhat diminished consistence, but otherwise normal.

The larynx and bronchi contained black flakes, which had evidently passed in from the gullet; their mucous membrane was pale. Extensive,

[1] By *saturation* is meant effervescing draughts, prepared by adding citric and tartaric acids, vinegar, &c., to the various alkaline carbonates.—Transl.

thin ecchymoses were observed beneath the pleuræ on both sides; there were likewise smaller ecchymoses in the mediastinum and pericardium, and beneath the epicardium. The lungs were crepitant and congested throughout, and œdematous posteriorly. The heart was flabby, and contained no blood; its muscular tissue was softer than natural, and its valves were normal. There were numerous ecchymoses beneath the endocardium.

The stomach contained about three pounds of a fluid resembling coffee-grounds, which also filled the œsophagus, and extended far down into the small intestine; pale-yellow, solid masses of fæces were found in the large intestine. No loss of substance, or congestion worth mentioning was noticed anywhere in the mucous membrane of the stomach or intestine. The entire mesentery was covered with ecchymoses; the pancreas was flabby and congested. The spleen was small, of normal color and consistence. The kidneys were large, slightly jaundiced, and anæmic; their epithelium was fatty.

The liver was somewhat enlarged; its surface was smooth, and its margins rounded; several ecchymoses about the size of a groschen (6¼ lines) were observed beneath the serous covering both of the liver and of the gall-bladder. The consistence of the gland was somwhat softer than natural; its color was citron-yellow; the cut surface was very anæmic, and nowhere exhibited capillaries filled with blood; the lobules were very indistinct, and could only be made out by the darker-yellow color of the pigment at their centres. On microscopic examination, the place of the cells was found to be supplied by numberless large and small globules of oil and particles of coloring-matter.

The portal vein contained a small quantity of fluid blood. The lymphatic glands in the fissure of the liver were enlarged to the size of a hazel-nut. A small quantity of dark, viscid bile was found in the gall-bladder, which could be forced without difficulty into the duodenum.

The urine found in the bladder was free from albumen, and closely resembled that of Solinsky. (Obs. XVIII.)

The third case is a characteristic example of acute softening of the liver.

Observation No. XX.

Symptoms of Acute Gastric Catarrh, with great Fever.—Somnolence.—Coma.—Noisy delirium.—No tumefaction of the Spleen.—Jaundice.—Urine abounding in Tyrosine and Kreatine.—Death from Cerebral Paralysis.

Autopsy:—Softening of the Liver.—Disintegration of the Glandular cells, and commencing Atrophy.—Kidneys soft, and in a state of fatty degeneration.—Spleen of normal size, and congested.

A female, aged eighteen, strong, fully-developed, and who had previously enjoyed uninterrupted good health, was taken ill on the 6th of July, 1859, with symptoms of acute catarrh of the stomach, loss of appetite, headache, loaded tongue, &c. Four days after this, she was brought to All Saints' Hospital,—the severe febrile symptoms, and the condition of the sensorium, having raised a suspicion that she was laboring under typhus. On admission, the pulse rose to 120, and the patient soon pas-

ed into a state of deep coma, which at times was interrupted by noisy delirium. No roseolar eruption, however, nor splenic tumor, tenderness over the cæcum, diarrhœa, nor any of the other diagnostic characters of typhus could be made out. She was ordered to take Muriatic Acid.

On the 12th of July, jaundice made its appearance; the condition of the sensorium remained unchanged; the pulse was very irregular and fluctuated between 100 and 120; the skin became gradually yellow; bile-pigment could be detected in the urine, and the pultaceous stools became pale. Death occurred on the 14th, under symptoms of cerebral paralysis, stimulants having been administered without any benefit.

Autopsy, 10 hours after death.

A moderate degree of hyperæmia of the brain and its membranes.

The mucous membrane of the pharynx, œsophagus, stomach, and intestines was normal; the small intestine contained mucus slightly tinged with bile, and the large intestine, pale solid fæces.

The spleen was slightly enlarged,—6 inches long, 4 broad, and 1¼ inch thick,—and weighed 0.18 kilogramme (6⅓ ounces avoird.); its parenchyma was soft, and pale-red.

The kidneys were of normal size; their cortical substance was pale-yellow, soft, and tumid; the tortuous uriniferous tubes contained epithelium loaded with fat, and for the most part were undergoing disintegration. A cyst, the size of a cherry, with bloody contents, was found in the left ovary.

The most important changes were observed in the liver. This organ weighed 1.6 kilogr. (3 pounds 8⅓ ounces avoird.), and was thus somewhat atrophied, while at the same time its tissue was flabby, shrivelled (*welk*), and unusually soft, especially in the left lobe. The capsule was wrinkled and opaque, and underneath it, in the deep-yellow, glandular substance were seen extravasations of blood the size of a linseed. On section of the organ, softened parts could be distinguished, of an irregular form, and of a pale-yellow or reddish-brown color, from which every trace of the outline of the lobules had disappeared, although in the adjoining, firmer portions, they were still quite distinct. The softening appeared to follow the ramifications of the portal vein, and the hepatic cells in the softened parts were destroyed and converted into a granular débris, oil-globules, and pigment molecules; whereas in the firmer portions of the right lobe, entire cells loaded with fine granules could still be distinguished. The portal and hepatic veins contained no blood, and in the hepatic artery there was nothing abnormal. The bile-ducts were pervious, and moistened with a pale-yellow mucus; the gall-bladder contained only a few drachms of greenish-brown secretion, which yielded no pigment when treated with chloroform. A gray granular efflorescence of tyrosine crystals was formed after some hours upon the cut surface of slices of the liver, allowed to dry. On chemical examination of the hepatic parenchyma, large quantities of leucine and tyrosine, and likewise of kreatine and kreatinine were detected; the compound of the last of these substances with chloride of zinc was obtained. The existence of both the last-mentioned bodies was proved by Liebig's process for examining muscular tissue.

The urine drawn off by catheter during life had a specific gravity of 1020, and a very acid reaction, and contained no albumen; when treated with chloroform, it yielded a moderate quantity of hæmatoidine. Crys-

tals of tyrosine, kreatine, and kreatinine remained, after evaporation of a small quantity of the urine upon an object-glass. On further examination, these substances were obtained pure, in large quantities. Leucine could not be discovered in the crystalline form, until after repeated treatment with alcohol. No urea could be detected.

The urine taken from the dead body was acid, pale, and no longer yielded any pigment when treated with chloroform.

It is remarkable that no enlargement of the spleen was present in any of the three cases just communicated.

The unfavorable nature of the prognosis in this affection has been mentioned already, under the head of Acute Atrophy; perfectly developed cases always terminate fatally. Earlier stages, however, of the inflammatory process appear, under certain circumstances, to undergo resolution, so as to allow of a cure. The following observations seem to favor this view:—

Observation No. XXI.

Fifth month of Pregnancy.—Bilious Vomiting.—Constipation.—Violent Headache, increasing so as to cause loss of consciousness.—Enlarged and painful Liver.—Tumefaction of the Spleen.—Albuminuria.—Slight Jaundice.—Cure.

Christiane Wels, aged 40, a tailor's wife, in the fifth month of her fourth pregnancy, was admitted on the 8th of July, 1858. Her disease commenced fourteen days before, with violent headache, giddiness, great faintness, and slight anorexia. On the 4th, she had a severe rigor, followed by persistent heat; on the 5th, she had repeated vomiting of bilious matter and an increase of the headache, amounting to loss of consciousness (*Unbesinnlichkeit*). Meningitis was diagnosed; leeches were applied, and calomel was administered, without producing any action of the bowels.

On admission, consciousness unimpaired (*volle Besinnung*); violent headache; pale countenance; pulse 120; heart's sounds and respiration normal. The right hypochondrium and epigastrium were tense and very tender; the volume of the liver was somewhat increased, its dulness in the sternal line amounting to 5, in the mammary line to 9, and in the axillary line to 10 centimètres (2, 3½, and 4 English inches); a soft splenic tumor was perceptible. The urine was scanty, turbid from the presence of lithates, but free from albumen and bile-pigment. Phosphoric Acid and Infusion of Senna were prescribed.

On the 10th, pulse 120; respirations 42. Several thin, grayish-yellow stools containing very little bile; urine very scanty and albuminous; the pains in the right hypochondrium continued, and the hepatic dulness was unchanged; the face exhibited a slight jaundiced tint. Tincture of Colocynth was ordered in addition to the Phosphoric Acid.

On the 11th, pulse 112; respirations 42. The incomplete loss of consciousness (*Eingenommenheit des Kopfes*), and the headache had abated; hepatic region still painful; hepatic dulness 6 centimètres in the mammary line, and 2 in the sternal (2⅓ and ¾ English inches); the spleen was likewise reduced in size; the urine still contained albumen, and deposited a gelatinous mucous sediment; leucine could not be discovered in the urine. Was ordered to continue taking the same medicines.

On the 12th, pulse 84; the hepatic region free from pain; the stools loaded with bile; the urine was free from albumen, and deposited a copious sediment of uric acid. Return of the appetite. Distinct fœtal movements.

From this time, the woman recovered with tolerable rapidity, and on the 19th of July, she was able to be discharged.

In the summer of 1859, I made a similar observation in the case of a young man, aged 16, who came under treatment for slight jaundice and a painful enlargement of the liver. The patient was drowsy and delirious at night; enlargement of the spleen, slight albuminuria, epistaxis, petechiæ, and other symptoms of disintegration of the hepatic cells were likewise present. On the administration of purgatives, and subsequently of acids, these symptoms ceased, so that about the eleventh day a complete cure was effected.

Oppolzer (*Deutsche Klinik*, No. 28, 1859) met with a favorable result under similar circumstances, although in his case the process had advanced so far, that leucine and tyrosine were present in the urine.

As regards the symptoms, diagnosis, etiology, and treatment of this form of inflammation, I must refer to the observations already made in the first volume (pp. 138 *et seq.*). I shall merely observe at present, that the diminution in the size of the liver, which was found to exist in cases I. and II., and also the splenic tumor, must not be looked upon as constant symptoms of diffuse inflammation.

DIFFUSE INFLAMMATION OF THE LIVER.

b. The Chronic Form.

(*Hepatitis Diffusa Chronica Adhæsiva.*)

The Simple and the Granular Induration of the Liver.—Cirrhosis of the Liver.—Interstitial Hepatitis.—Hob-nailed Liver.—Gin-drinker's Liver.

1. *Historical Account.*

Induration of the liver, as a consequence of inflammation, was known to the ancients,[1] and numerous descriptions of it are found in old pathological works, under the designations of *hepar durum*, *subdurum*, *subcultro stridens*, *scirrhus*, and *obstructio hepatis*, *marasmus hepatis*,[2] &c.; many cases of this nature were published by Morgagni.[3] Very different lesions of the liver, however, were included under these names; and even Morgagni did not distinguish between carcinoma and simple induration.

The granular induration was likewise observed at an early period, and was well described by many writers. Vesalius[4] records the case of a

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[1] ARETAEUS (*De causis et signis morborum diuturn*, Lib. I., Cap. xiii.) observes: Verum si a phlegmone hepar non suppuratur, nemini dubium fuerit, tumorem durum subsidentem in scirrhum mutari ac stabiliri.

[2] BIANCHI (*op. cit.* pp. 401 to 403) describes the following condition under the name of *Marasmus hepatis*:—"Jecus deprehenditur prorsus aridum, coriaceum, in minimam molem retractum."

[3] MORGAGNI, Epist. XXXV., 2, 4, 23, 25; XXVIII., 16, 20, 30, &c.

[4] VESALIUS (*Opera*, Tom. II., p. 674).

lawyer, who, after having suffered for a long time from symptoms of obstruction of the liver, died suddenly when sitting at table. On examining the body, the trunk of the portal vein was found torn, the abdominal cavity filled with blood, and "hepar totum candidum et multis tuberculis asperum, tota anterior jecoris pars et universa sinistra sedes instar lapidis indurata erat." Nicol. Tulpius,[1] on opening the body of a man, who had suffered from ascites and tympanites, and who had passed blood upwards and downwards, found the spleen enlarged, and the liver "aridum et retorridum." In the body of a jaundiced and dropsical female also,[2] the liver was: "(jecur) aridum, atrum, exsuccum et instar corrugati corii in se contractum, ut vix æquaret geminum pugnum." Morgagni, in his thirty-eighth Epistle, details a series of observations, partly his own, and partly borrowed from Posth, Wepfer, and Ruysch, which were undoubtedly instances of cirrhosis. Thus, he found in the body of a Venetian noble, whose case is given in detail: "jecur durum, intus extraque totum constans ex tuberculis, id est glandulosis lobulis evidentissimis, et evidentissime distinctis, nec tamen naturali major." Posth,[3] in a case of ascites, described the liver as: "totum granulosum, granis nimirum quantitate pisorum ubique apparentibus." Morgagni had a clearer idea of cirrhosis than many more recent writers. He rightly observed: "non possunt minimæ jecinoris partes adeo amplificari, quin aut interjectas alias, aut vascula saltem sanguifera comprimendo, hepatis muneri et sanguinis perventrem motui plurimum officiant."

The term "tubercles," by which Morgagni designated the prominences of the liver, has since his time been variously misused. Matthew Baillie[4] employed this term not only in reference to the granular induration of the liver, but also in speaking of cancer; and Meckel[5] committed a similar mistake.

Laennec was the first who called the morbid change under consideration, cirrhosis (κιρρός), and who enunciated the view that the nodules were to be regarded as new formations, which might be developed in other organs as well as in the liver, and which, like other new formations, might undergo softening.

Notwithstanding the great authority of Laennec, this view soon met with several opponents, before whom it was obliged to succumb. In the year 1826, Bouilland[6] endeavored to show, that no new formation existed in the lesion in question, and that the yellow granulations consisted of the glandular parenchyma, which gradually passed into a state of disorganization, owing to disease of the vascular connecting tissue. This opinion was supported in the main by Andral,[7] except that he bestowed a greater degree of prominence upon the distinction between the red vascular portion, and the yellow secreting tissue of the gland, and looked upon the granulations as an hypertrophy of the latter, accompanied by atrophy of the red vascular portion, which in many cases appeared to be converted into fibrous tissue. Cruveilhier[8] disputed the existence of two different substances in the liver, and looked upon cirrhosis as the result of atrophy

[1] NICOL. TULPIUS (*Obs. med.*, Lib. II., Cap. XXXV., p. 153).
[2] *Ibid*, Cap. XXXVI., p. 154.
[3] MORGAGNI (*loc. cit.*, Epist. XXXVIII).
[4] BAILLIE: Pathological Anatomy.
[5] MECKEL: Path. Anat., Part. II., p. 318.
[6] BOUILLAND: *Mém. de la Société méd. d'Émul.*, Tom. IX., 1826.
[7] ANDRAL: *Précis d'Anat. Pathol.*, Tom. II., p. 853. Paris, 1829.
[8] CRUVEILHIER: *Anat. Pathol.*, (Ed. 1856. Tom. III., p. 216.)

of one portion of the gland, with hypertrophy of the remaining portion. Becquerel[1] constructed a new theory, according to which the so-called yellow substance was the peculiar seat of the disease in cirrhosis; it was thought, that it became infiltrated with an albuminous material, and in this way hypertrophied, that the red vascular portion was compressed by it, and atrophied, and that at a more advanced stage the yellow substance itself was likewise atrophied.

The French observers arrived at no certain results, because they all proceeded upon indistinct views of the structure of the liver. An accurate knowledge of the lesion in question was first obtained through the investigations of Kiernan,[2] Hallmann[3] and Carswell,[4] who showed that an increase of the interlobular connecting tissue of the hepatic parenchyma was the essential feature of cirrhotic degeneration. Hallmann first drew attention to the frequent coexistence with this condition, of fatty degeneration of the hepatic cells. From this circumstance, Gluge[5] and Léréboullet[6] were led to the conclusion that cirrhosis proceeded from deposit of fat in the hepatic cells; Gluge, however, in addition to this form, which he designated "*Steatose*," described another which he believed to result from interlobular hepatitis.

Rokitansky[7] distinguishes two different modes of origin of hepatic granulation, the one proceeding from a morbid development of the capillary blood-vessels, owing to an excessive secretion of bile, the other due to a chronic inflammation of the hepatic parenchyma. Oppolzer[8] attributes cirrhosis of the liver mainly to partial impermeability of the finest ramifications of the portal vein, resulting from inflammation and obliteration, or from lateral compression by the bile-ducts, which are enlarged or loaded with fat.

The most recent authors, such as Gubler,[9] Budd,[10] Henoch, Bamberger, and others, all refer cirrhosis to a chronic inflammation of the liver, although their views vary in individual particulars.

2. *Anatomical Description.*

One rarely has the opportunity of tracing the development of induration of the liver during life, or of examining anatomically the early stages of the lesion; as a rule, the disease only comes under observation when it is more or less completely developed, and when the consecutive disorders draw attention to the organ primarily diseased. Hence in most cases, cirrhosis and induration can only be inferred during life by tracing backwards the clinical history of the cases where these morbid changes are found after death.

For this reason, we shall consider, in the first place, the anatomical characters of granular induration of the liver, in order to obtain reliable

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[1] BECQUEREL: *Archiv Gén. de Méd.* 1830.
[2] KIERNAN: *Philosoph. Transact.* 1833.
[3] HALLMANN: *De cirrhosi hepatis.* Berol., 1839.
[4] CARSWELL: *Pathol. Anatomy. Atrophy.*
[5] GLUGE: *Atlas des Patholog. Anatomie.*
[6] LÉRÉBOULLET: *Mémoires prés. à l'Acad. des Sciences.* 1851.
[7] ROKITANSKY: *Patholog. Anatom.* Bd. III., S. 334.
[8] OPPOLZER: *Prager Vierteljahrsschrift.* Bd. III., S. 17.
[9] GUBLER: *De la Cirrhose, Concours par l'Aggrégation.* Paris, 1853.
[10] BUDD: *Diseases of the Liver*, 2nd Ed., p. 134.

data for the clinical elucidation of the origin, causes, consequences, and symptoms of the disease.

The vascular apparatus and secreting structures of the liver are supported by a framework of connective (areolar) tissue, which pervades the entire organ and bounds it externally. In the first place, beneath the peritoneal covering there is a capsule of connective tissue containing a large number of elastic fibres, which envelopes the gland; in addition to this, Glisson's capsule, consisting of the same elements, penetrates at the fissure of the liver into the interior of the gland, and accompanies the vessels, nerves, and bile-ducts, as far as their finest ramifications. There is likewise an alveolar matrix of an amorphous connective substance in the meshes of which lie the hepatic cells, whilst the capillaries are dispersed through the walls.

The areolar framework of the liver just described may become hypertrophied, and the farther this process proceeds, the more is the consistence of the organ increased. On examining the consistence of the liver after death, we find important differences, the discrimination of which requires a certain amount of experience. These differences are due, partly to the nature of the cell-contents (the gland being softer when the cells are loaded with fat, and firmer when they contain amyloid matter), and partly to the amount of blood and serous infiltration contained in the gland, but mainly to the degree of development of the areolar framework. When this is increased, the organ is not merely firmer, but also more tenacious. This increased consistence is particularly frequent in persons who have been addicted to the use of spirits; it is also observed to result from derangements of digestion, and from persistent intermittent fever, and sometimes there is no obvious cause to account for it. The gland, in this state, may appear to the naked eye unchanged, or the lobules may be unusually distinct and sharply defined, whilst the capsule appears opaque and thickened.

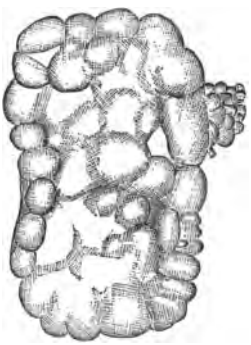

FIG. 5.—represents a liver in a state of cirrhotic degeneration. The left lobe is particularly atrophied; while the right is, at the same time, enlarged from amyloid deposit.

These cases of increased consistence of the liver constitute the commencement of cirrhotic degeneration; between them and the most advanced forms we find numerous intermediate grades, which gradually pass into one another, and which may be all regarded as different stages of the same morbid process. Here, only the two extremes—corresponding to the commencement and termination of this process of disorganization—are described.

In the slighter forms of granular induration, the liver is somewhat enlarged, or of normal size, rarely smaller than natural; its surface is covered by a smooth, or somewhat opaque, and thickened capsule, and exhibits flattened projections varying in size from a pin's head to a pea; the external contour of the organ is little altered. Nodules, similar to those on the outer surface, are observed in the interior; they are here separated from one another by narrow strips of a gray, or more or less vascular, areolar tissue, and usually present the normal reddish-brown tint; in rare cases, the color is darker from the presence of bile-pigment, or paler, in consequence of the deposit of fat.

The advanced form of the disease differs from this in many respects. The liver is then reduced in size, and particularly the left lobe, which not

unfrequently is shrivelled up into a small membranous appendage, and, in consequence of the atrophy, there is a soft flabby rim of connective tissue at the margins of the organ. Semi-globular knobs (Höcker), more or less prominent, sometimes of a uniform, and at other times of an unequal size and form, are thickly scattered over the surface. The serous envelope is almost always thickened and coriaceous, and of a grayish-white color, especially in the depressions between the granulations; numerous bands of connective tissue pass from it to the neighboring organs, such as the diaphragm, colon, stomach, &c. On section, the organ presents a cartilaginous hardness and coriaceous tenacity, and we observe at one place narrow, and at another broad lines of connective tissue, of a gray color, surrounding the granulations, and sending streak-like processes into their interior. These granulations are in most cases dark- or pale-yellow, rarely greenish, brown, or reddish.[1]

In order to obtain a deeper insight into the structural changes which the liver undergoes in cirrhotic degeneration, it is necessary to trace more closely the characters of the individual elementary parts; in this way we shall at the same time obtain some information as to the functional derangements of the organ.

a. The secreting substance of the gland and the granulations.—A large portion of the hepatic cells are destroyed; their remains are found in the form of small masses of brownish pigment scattered through the filaments of the newly-formed connective tissue. Another portion of them constitutes the substance of the granulations, and may remain for a long period intact. In the further progress of the disease, these cells likewise usually undergo changes, which interfere with their functions to a greater or less extent; the cells become filled with fat and with various sorts of pigment. I have found fatty degeneration of an advanced stage, in nearly one-half of the cases of cirrhosis which have come under my observation; in most cases, it is to be regarded as the consequence of the deranged nutrition of the gland, produced by the chronic inflammation, as is evident from the fact, that not unfrequently some of the small granulations, which are surrounded by thick fibrous bands, are in a state of fatty degeneration, while others remain normal. In many cases, however, the cirrhotic degeneration appears to take place in a liver previously fatty.

The deposit of pigment, from which the term cirrhosis is derived, although not always very remarkable, is seldom absent, because the connective tissue compresses, to some extent, the commencement of the bile-ducts, and thus gives rise to a retention of the secretion, and a jaundiced condition of the liver. The coloring-matter accumulates in the form of a fine, orange- or sulphur-yellow granules; more rarely it is diffused through the cavity of the cells. Owing to its presence, the granulations in most cases appear of a deep yellow color, although occasionally they are olive-green, or rust-brown. Besides this color, which is due to the presence of bile-pigment, another tint may occur, arising from the decomposition of the red matter of the blood. I have seen this in an extensive form accompanying obliteration of the branches of the hepatic veins. In this case, the veins were observed to be infiltrated with dirty red, brown and black pigments, while, at the same time, the remains of the vessels entering the lobules were observed to be filled with stagnant and similarly-colored blood.

Of much rarer occurrence than the deposit of fat or pigment, is a third

[1] For the relative size and weight of the cirrhotic liver, see Vol. I., pp. 20, 21.

pathological change, viz.: the lardaceous or amyloid infiltration. The granulations, in this case, present the same characters as the parenchyma of the lardaceous or amyloid liver; they are firm, dense, and of a waxy lustre, while a large number of the cells are filled with an amorphous substance of the ordinary reaction, and others contain fat or pigment.

Under such circumstances, the liver, even in an advanced stage of cirrhotic degeneration, exceeds its normal size. (Observation No. XXXIV.)

b. The connective tissue.—The increased amount of connective tissue in the liver presents numerous varieties, as regards its mode of distribution, to which are due the differences in the size of the granulations. The increase is usually first distinguishable in the prolongations of Glisson's capsule,[1] which accompany the finer subdivisions of the vessels in the interior of the liver; from this it gradually proceeds to involve the substance of the lobules. The fibrous bands at one time enclose single acini, and at another, three, five, or even a larger number. Increasing in breadth, these bands completely destroy, by their pressure, individual lobules, so that a little mass of brown pigment is the sole trace remaining of them. Now and then, we observe the hepatic parenchyma destroyed in this way over a great extent, and its place supplied by a mass of connective tissue.

In a similar manner to what takes place in the case of Glisson's capsule, although to a more limited extent, the connective substance passes from the serous envelope inwards into the interior of the liver, compressing and destroying the glandular elements. (Observation No. XXIV.)

The newly-formed tissue does not at every part present the sama elementary characters. At the circumference of the lobules we find fibrillated connective tissue, which always contains elastic fibres in the neighborhood of the larger branches of the vessels. In the substance of the lobules, on the other hand, the connective tissue has an amorphous character, and in the thickened capsule it is fibro-cartilaginous.

c. The vascular apparatus of the liver always undergoes remarkable and important changes. The trunk and larger branches of the portal vein usually remain unchanged, but the smaller subdivisions are in most cases narrowed by the shrivelling of the connective tissue; they lose their rounded form, and become angular and bulging (*buchtig*). Sometimes the trunk and branches are enlarged and filled with old clots. In three cases, Carswell observed the portal vein enormously distended, and filled, as far as its finer ramifications, with a firm mass composed of coagulated blood, fibrine and bile; in these cases the circulation must have ceased a long time previous to death. Monneret also describes a case where the vena porta and the hepatic veins, as far as their opening into the vena cava, and likewise the bile-ducts, were enormously enlarged; he also records another case, in which the main branches of the portal vein were found blocked up. This formation of a thrombus may be accounted for by the destruction of a large portion of the capillaries, and the consequent obstruction to the circulation.

The changes, which take place in the capillaries of the portal vein, are more constant than those observed in its trunk and large branches; the former are destroyed to an extent proportioned to the disappearance of the glandular substance of the liver. So long as the hepatic cells are still visible, we can succeed, by means of injection, in demonstrating the peculiar mesh-like capillary network of the liver; but where these cells have

[1] The finer prolongations of this capsule are here referred to: the sheath of the larger branches of the vessels is often found thickened without any trace of granulation.

disappeared and their place is supplied by connective tissue, entirely new capillary channels everywhere make their appearance, which form elongated meshes, and may be injected not only from the veins but also from the hepatic artery.[1]

Thus new channels are developed, passing from the portal vein into the hepatic veins; but their number is limited, and, in most cases, insufficient for transmitting the blood contained in the portal vein.

Important alterations may likewise be distinguished in the hepatic artery. Its trunk becomes enlarged,[2] and its capillary network is much more extensive than in the healthy state: I have usually found a quantity of black pigment accumulated in some of its branches. When the hepatic artery is injected, there appears in the connective tissue a very much branched and tortuous net work of vessels of comparatively large calibre, the mode of distribution of which shows clearly their new formation.

The hepatic veins, as a rule, present nothing abnormal; in one case only have I observed several branches of this vessel obliterated, and, as a consequence of this, numerous apoplectic masses and bloody suffusions in the substance of the liver; the closure of the hepatic veins was caused by the propagation of inflammation from the capsule of the liver to the walls of the vessels.

The capillaries of the hepatic veins are gradually destroyed, and then their communication with the portal capillaries is interrupted; on injecting the hepatic vein, extravasations take place in the substance of the granulations, and none of the injected matter penetrates into the portal vein.

d. The bile-ducts.—The origin of the bile-ducts at the periphery of the lobules is, as already stated, partly destroyed by the pressure of the newly-formed connective tissue; not unfrequently, there is catarrhal tumefaction of the mucous membrane of the larger branches; in other respects, the ducts usually present nothing abnormal.[3] The walls of the gall-bladder are often thickened and adherent to the neighboring parts; its contents are, in most cases, scanty, liquid, and of a pale- or orange yellow color.

The above are the most important changes, which the liver undergoes in cirrhotic degeneration. They give rise to a long series of functional derangements, which, in practice, constitute the symptoms of cirrhosis. The principal of these are the following:—

1. Functional derangements of the chylopoietic organ, arising from an impediment to the passage of blood from the portal into the hepatic veins, and its stagnation in the roots of the portal vein.

2. Impairment, passing on to complete suspension, of the hepatic functions.

3. An impairment of those functions, which, in addition to the secretion of bile, the hepatic parenchyma performs in the metamorphosis of matter and in the elaboration of the blood.

3. *Etiology.*

The degeneration of the liver which has just been described, has in recent times been pretty generally attributed to a chronic inflammation

[1] The capillaries of both the artery and the vein become filled, whichever vessel is injected.

[2] I have repeatedly found the circumference of the hepatic artery, in the fissure of the liver, measuring from 14 to 15·5 millimetres (6¾ to 7¼ English lines).

[3] Gubler found the ductus hepaticus enlarged and filled with stagnant bile, and he attributed this enlargement, like that of the bronchi in chronic pneumonia, to a retraction of the new connective tissue.

of the gland, in the same way as the development of connective tissue in other organs and tissues, where the injection of the vessels and the increased effusion of plasma can be directly observed, is referred to a like source. As regards the liver, it is usually impossible to do this; it is rare, especially in hospital practice, that we meet with cases where we can trace the process clinically from its commencement to its termination in complete degeneration. On the *post-mortem* table, it is true, we may often succeed in observing the early stages of granular induration, and then, indeed, the hyperæmic enlargement of the gland favors the idea of an inflammatory derangement of nutrition. But one may justly ask, Is granular degeneration of the liver always preceded by this stage? Has this morbid condition always the same origin; or may it not arise from various morbid processes?

There can be no doubt that granulations of the liver may arise in other ways, and, so far, the theories which call in question their inflammatory origin, have a certain amount of truth; but there is only a superficial resemblance between such granulations and those of true cirrhosis. Granulations of this nature are met with under the following circumstances:—

1. *In fatty liver.*—In this affection, segments of the liver, in which the cells are extraordinarily distended with fat, project in the form of yellow, rounded granulations, the size of a pin's head. In all cases of this nature which I have examined, the prominences have been formed by the portal zones of the lobules, and, after injection, twigs of the portal vein and hepatic artery might be seen on the top of the granulations, whilst the hepatic veins lay in the depressions. In true cirrhosis, the relation of the parts is precisely reversed; the hypertrophy of the connective tissue, the development of new vascular channels and the great firmness and tenacity of the gland, which always characterize genuine cirrhosis, are completely wanting in fatty liver, which is likewise exempt from any marked derangements of the circulation.

2. *In consequence of hyperæmias from obstructed circulation in cardiac and pulmonary diseases.*—Here, also, the gland becomes finely-granular, and at the same time firmer and more tenacious, and a condition is developed, which has frequently, and particularly by Becquerel, been mistaken for cirrhosis. The granulations, however, are formed in an entirely different manner; the roots of the hepatic veins are enlarged as far as their capillary origin, and cause the surrounding cells to disappear. In this way, the parts corresponding to the hepatic veins sink down, whilst those occupied by the portal veins project as fine granulations. At first, the atrophy is confined to the circumference of the enlarged capillaries, but, after a time, it extends to the large branches, so as to produce extensive depressions. At the same time, the walls of the vessels, being subjected to an abnormal degree of pressure, usually become thickened, new connective tissue is developed around the vessels, and occasionally likewise in the capsule of the gland, and this imparts to the organ a greater degree of firmness.

3. *In consequence of pyle-phlebitis adhæsiva.*—Obliteration of the fine branches of the portal vein is, as a rule, followed by atrophy of the surrounding hepatic substance; depressions are formed, which give to the liver a lobulated, and sometimes a coarse granular, aspect. This condition, however, differs essentially from true cirrhosis; the greater size, the less regular form, and the flatter character of the projections, as well as the absence of the bands of connective tissue in the interior of the liver,

distinguish this form of atrophy, at first sight, from that arising from cirrhosis; on tracing the branches of the portal vein into the interior of the liver, we arrive at blind extremities, which are surrounded by the firm, shrivelled, hepatic tissue, whilst the remaining portion of the parenchyma is still unchanged. (Vide *Pyle-phlebitis.*)

Oppolzer's views (see p. 70) concerning cirrhosis of the liver, apply to cases of this nature, and not to the real granular induration.

4. Rokitansky refers one form of cirrhosis to *a morbid development of the capillary bile-ducts.* I have never met with cases which would admit of such an explanation. In the various grades and forms of obstruction to the flow of bile, which have come under my notice, the liver never presented any granular character. The enlargement of the bile-ducts did not extend to their origin, but apparently terminated earlier, in the form of elongated hollow spaces, surrounded by hepatic cells loaded with pigment.[1]

Thus we regard the various conditions just described as essentially different from cirrhotic induration, which we refer to a chronic interstitial hepatitis (*Hepatitis interstitialis*).

The causes of this interstitial hepatitis are, in part, at least, well known. The chief of these causes is *the abuse of spirituous liquors*, which is regarded by all observers[2] as the ordinary exciting cause of cirrhosis; hence the English apply to this disease the term "gin-drinker's liver."

Of 36 cases of cirrhosis which have come under my observation, twelve of the patients confessed to having been in the habit of drinking brandy in excess, and several of the others were suspected of the same vicious habit.

In the maritime towns of Northern Germany and England, where strong spirits are frequently drunk in excess, cirrhosis is more prevalent than it is in the interior of those countries, where the use of beer or wine is more common. I have met with cirrhosis and delirium tremens far more frequently at Kiel than at Göttingen or Breslau. Alcohol, in its least diluted form, is particularly injurious to the liver; wine and beer, which contain a varying amount of water, in addition to the spirit, appear, so far as my experience goes, not to produce this effect. The rapid absorption of the spirits from the stomach into the portal vein, must in the first place give rise to irritation of the liver, which after a time subsides, the more that the absorbed fluid becomes mixed with the entire mass of blood, and evaporated through the lungs.

After poisoning animals with alcohol, Percy found the largest quantity of alcohol in the liver. Budd justly observes, that spirits are most injurious when they are taken neat, that is, undiluted with water, and on an empty stomach; the latter condition is particularly favorable to their rapid absorption and to their powerful action upon the liver.

Whether there are other acrid ingesta, besides alcohol, which by being transmitted in the portal blood through the liver, can irritate this organ in such a manner as to give rise to chronic inflammation with subsequent induration, is a question which as yet has not received a satisfactory answer. Budd is inclined to ascribe the frequent occurrence of cirrhosis in

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[1] See Vol. I., Fig. 20, pp. 82 and 98.

[2] Long ago, Vesalius (*De humani corporis fabrica*, Lib. V., p. 507) stated, that there was a prevalent opinion among anatomists, that the liver was reduced in size by drinking:—"Insignibus illis gurgitibus vini jecur ad nucis duntaxat volumen reduci."

India to the excessive use of curry and other irritating spices, and there can be no doubt, as has already been stated (p. 43), that these and similar substances, such as strong coffee, may excite transient hyperæmias of the liver.

In a large number of cases, however, we are unable to attribute the disease to the abuse of spirits, or to any other irritating articles of diet, and then it is a more difficult matter to trace the origin of the complaint. Very often we are unable to discover any exciting cause, or we merely obtain more or less obscure references to previous disease of the liver.

Of the 36 patients who have come under my observation, six were suffering from constitutional syphilis, or had formerly presented the symptoms of this affection. In three of these cases, the hepatic parenchyma was at the same time infiltrated with colloid matter, which was likewise found in the spleen and kidneys. I think, therefore, we may assume a connection between the syphilitic dyscrasia and cirrhosis of the liver, and the more so, as other deranged conditions in the nutrition of the liver, such as fatty or colloid degeneration, circumscribed inflammations, &c., may also result from syphilis.[1]

In five patients the cirrhosis was preceded by persistent intermittent fever — a coincidence which has likewise been repeatedly mentioned by other observers. On the whole, however, granular induration of the liver is a rare lesion in individuals who have died from the cachexia of intermittent fever, and I have more frequently found either simple chronic atrophy or fatty, and occasionally, colloid infiltration. It would appear, therefore, that other agencies must co-operate, when cirrhosis comes to be developed under such circumstances as those just mentioned; but in the meantime, the precise nature of these agencies remains obscure.

The derangements in the circulation of the liver which have been observed to result from diseases of the heart, have frequently been regarded as a cause of cirrhosis. Of 42 cases of cirrhosis, Becquerel found disease of the heart in 21; in 13 of these, however, the condition of the organ was what he designates the first stage of cirrhosis, and was attended either by no symptoms at all, or by none of a marked character. But, as we have already seen, the condition which the organ assumes under such circumstances is essentially different from cirrhotic induration, and hence the value of Becquerel's observations is, in my opinion, much impaired. It is true that diseases of the heart are found accompanying true cirrhosis, as I have noticed in 4 out of 36 cases, but they are not the cause of the degeneration of the liver, but rather complications which modify its symptoms, and hasten its fatal termination.

There are, undoubtedly, causes of cirrhosis with which we are as yet totally unacquainted. I have observed this lesion in a boy, ten years of age, who had not been subjected to any of the injurious agencies just mentioned; Rilliet and Barthez have likewise met with it in children, and F. Weber[2] has even seen it in newly-born infants as a congenital disease transmitted from the fœtal period of life.

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[1] For the anatomical differences between cirrhosis resulting from syphilis, and that resulting from the abuse of spirits, see farther on.

[2] F. Weber, *Beiträge zur pathologischen Anatomie der Neugeborenen.* 3 Lief. S. 47. Kiel, 1854.

One child, of a twin birth, came into the world dead, whilst the other was healthy. The former was emaciated, jaundiced, and covered with petechiæ; the abdominal cavity contained a considerable quantity of yellow serum; the mucous membrane of the stomach was pale, that of the large intestine injected and tumid. The most important

As regards the general predisposing causes of age and sex, 20 of my 36 cases were males, and 16 females; the number of females, however, may be too large, because of the women who are admitted into public hospitals far more are addicted to drink and other excesses than is usually the case with this sex. In reference to age, the cases were distributed as follows:—

From	10	to 20	years,	1 case.
"	20	" 30	"	2 cases.
"	30	" 50	"	12 "
"	50	" 70	"	20 "

In addition to these 35 cases, there was a female affected with cirrhosis, whose age was 81.

These numbers, however, are far from being sufficiently large to determine the influence of age with any degree of accuracy.

4. *Symptoms of Cirrhosis.*

A. *General clinical history of the disease.*

The disease is usually developed slowly and insidiously; it is only in exceptional cases, where the primary inflammation is acute and implicates the capsule, that the commencement of the process is distinctly announced by derangements of a definite character. Dull pains in the right hypochondrium are then complained of; the region corresponding to the liver is tense and distended, and the size of the organ is increased. At the same time, we may notice a slight degree of fever, accompanied by derangement of the stomach, loaded tongue, nausea, and, occasionally, vomiting and faint jaundice. After a longer or shorter duration, these symptoms most commonly abate, although the degenerative process in the liver advances and gradually undermines the constitution. Not unfrequently, the symptoms at first are so trifling, that they are passed over unnoticed, and only attract attention when the disease is far advanced.

The patients complain of feeble digestion, which is disordered by articles of diet that formerly could be taken with impunity; their appetite falls off; distention and tenderness of the epigastrium along with flatulence and constipation are developed; and, after a time, these symptoms abate, but return from some slight exciting cause. By degrees the patients lose flesh and strength; their color becomes pale, or earth-colored, or sometimes dirty-yellow, while their skin is dry and rough. At the same time, the abdomen becomes distended and fluctuates; and on closer examination, the liver is found reduced, and the spleen, as a rule, increased in size. The pulse continues of normal frequency, but gradually becomes softer; while the ascites and tympanites occasion more or less dyspnœa. The appetite, which at the commencement is in most cases impaired, not unfrequently returns at a later date, but does not arrest the patient's de-

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morbid change was found in the liver, which was small, greenish-brown, and very granular; broad bands of connective tissue separated the islets of parenchyma, which projected from the surface in the form of knobs, and which were of a deep jaundiced tint. It is worth noticing, that the sinuses of the brain were remarkably enlarged, and filled with blood.

cline; in place of constipation, the bowels become regular, the stools being for the most part pale, or in some cases there is diarrhœa. Occasionally, hæmorrhages from the stomach or intestine take place. The urine, which at first is tolerably abundant, becomes scanty, as the dropsy advances; and, at the same time, assumes a deep red hue, is often turbid, and deposits a brick-red sediment, but rarely presents a jaundiced tint.

As the disease progresses, the emaciation and debility increase; slight febrile excitement manifests itself; the appetite fails completely; the dyspnœa becomes more urgent from the increasing distention of the abdomen, and in most cases diarrhœa supervenes, which terminates in death from exhaustion.

In other cases, the fatal termination is induced by pneumonia, acute pulmonary œdema, or peritonitis; occasionally it occurs under symptoms of acholia; the patients become jaundiced, purpura spots or ecchymoses are scattered over the skin, and delirium, convulsions, and, finally, deep coma close the scene.

Such is a general sketch of the main clinical features, under which granular induration of the liver presents itself to us in practice. We must now fill in the details, by considering more closely the individual symptoms in reference to their origin, pathological importance and diagnostic value. We shall confine ourselves in the first place to the local symptoms, in so far as they concern the liver and the abdominal organs connected with it.

B. *Local Symptoms.*

a. *Characters presented by the liver.*

In rare instances, the cirrhotic liver is found enlarged, either because the gland is in the early stage of degeneration, or because it is infiltrated with amyloid matter. In the former case, the incipient stage of the process can only be recognized by palpation, when the gland is tender upon pressure and granulations can be felt over the surface; in the latter case, the nature of the lesion is detected more easily by means of palpation. With a little practice, the surface of the organ is then felt to be granular on pressing the point of the finger against it, and the granulations are easily distinguished from the large nodules which are met with in carcinoma and lobulated liver (*Lappungen der Leber*); the firm consistence of the organ and the character of the margins, which are sometimes rounded and at other times sharp, also enable us to detect it without difficulty by means of careful palpation.[1]

In most cases, however, the liver is reduced in size, and is not easily accessible to palpation, especially where there is a considerable amount of ascites, when the gland is covered by folds of intestine containing gas, or when its margin is tilted up and pushed high into the hollow of the diaphragm. Under these circumstances, it is easy to ascertain by percussion that the dulness is diminished in extent, and that in the region of the left lobe it is, as is often the case, entirely absent; but it is a difficult matter to determine in this way the real size of the liver, inasmuch as the extent of the dull percussion sound may be very limited in ascites, even when there is no diminution in the volume of the organ, owing to its position

[1] In cases where there is still a thick layer of fat in the abdominal walls, every conceivable mode of palpation often yields no result.

being altered. Some degree of certainty can only be arrived at, when the examination is frequently repeated, or when there is an opportunity of determining the margins of the organ immediately after the performance of paracentesis of the abdomen. The smaller the liver and the greater the amount of the ascites, the more difficult it is to distinguish the granular surface and the hard, firm character of the margins—two points which, for the purpose of diagnosis, are of the utmost importance. This difficulty often continues throughout the whole course of the disease; but in other cases where the organ lies lower, or where, after paracentesis, the flabby abdominal walls permit the hand to penetrate farther, the observer may from palpation alone convince himself as to the nature of the lesion, and form an accurate diagnosis.[1]

The hepatic region usually exhibits no tenderness during the examination; in most cases, the right hypochondrium feels soft and is free from tension; it is only at the very commencement of the disease, or for brief periods during its further progress, when there are aggravations of the peri-hepatitis, that the hepatic region is tender, and then there may be also dull pinching pains coming on spontaneously, but which never attain great severity.

From what has been stated, it is evident that direct examination of the diseased organ rarely furnishes data sufficient for arriving at a correct diagnosis. Very often such an examination proves nothing more than a diminution in the volume of the gland, and even this may be doubtful, when there is a considerable amount of ascites with persistent tympanites of the bowel.

Hence it becomes the more necessary to pay particular attention to those functional derangements which result from granular induration, and which impart to the disease that peculiar character, from which its existence may be recognized.

The most of these derangements are referrible to the obstruction of the circulation in the portal vein; others depend upon the impaired functional activity of the atrophied gland, while others result from the co-operation of the numerous consecutive morbid conditions which the disease of the gland entails.

The obstruction which the blood of the portal vein meets with in the liver has already been pointed out in the general description of cirrhosis; in a clinical sense, this affection was justly regarded by the ancients as an *obstructio hepatis.*

The new channels of communication, which are developed between the portal and hepatic veins (after a destruction of the capillaries of the portal vein, that varies in extent in different cases), being usually insufficient for the free circulation of the blood, a state of passive congestion arises in the portal system, which gives rise to a series of anatomical lesions and functional derangements. These are in general the more marked, the farther the granular induration of the gland has advanced, and the larger the number of the branches of the portal vein which have been destroyed; exceptional cases, however, do occur, owing to the development of collateral channels, which more or less completely compensate for the obstruction. Cases of advanced cirrhosis are observed, where derangements of the circulation are scarcely perceptible, and others are met with, in which the consequences of the obstruction disappear again completely with the pro-

[1] Bamberger errs in supposing, that the granulations can never be felt through the abdominal walls.

gress of the disease, owing to the obstruction being compensated for by the formation of new channels for the blood.[1]

Communications exist between the portal vein and vena cava, which, when enlarged, convey a large portion of the portal blood direct to the heart without passing through the liver. In the first place, there is the anastomosis of the internal hæmorrhoidal with the inferior hæmorrhoidal veins, which pass into the hypogastric vein, and likewise the anastomosis of the left coronary with the œsophageal and diaphragmatic veins. More rarely, we find anastomoses of the hæmorrhoidal with the vesical veins, of the coronary vein of the stomach and of the gastro-epiploic veins with the renal vein, of the superior mesenteric vein with the left renal vein, and of the vasa brevia with the left phrenic. There are few direct proofs, however, that these vessels become enlarged in cirrhosis. Fauvel is the only one who has found the œsophageal veins in a varicose state in this disease; and hæmorrhoidal varices, so far as my experience extends, are by no means frequent.

A second collateral channel for the blood of the portal vein is found in the newly-formed adhesions of the liver to the diaphragm and abdominal wall. These vessels pass into the diaphragmatic veins, and were recognized by Kiernan; I have succeeded in making them out in every case where I have injected the portal vein.

Of more importance than either of the above communications, are certain accessory branches of the portal vein described by Ph. Sappey,[2] which exist on the under surface of the diaphragm, and upon the inner surface of the epigastric portion of the abdominal wall, and which pass to the liver between the folds of the falciform ligament. One portion of these veins sinks into the convex surface of the gland, and here unites with the branches of the portal vein, while another winds round to the longitudinal fossa, and passes in at the under surface. The largest of these last-mentioned branches accompanies the ligamentum teres, and passes on to the left branch of the sinus venæ portarum. The roots of these veins send branches through the sheath of the rectus muscle, which anastomose with the epigastric and internal mammary veins, and partly, also, with the superficial abdominal veins. Sappey found these vessels considerably enlarged in cirrhotic degeneration of the liver; in two cases, the vein which accompanies the ligamentum teres was distended to the size of the little finger, and, in three other cases, to a less extent. These collateral channels have, hitherto been, for the most part, overlooked,[3] owing to the en-

[1] Monneret has recorded a case of cirrhosis, where the ascites disappeared completely after the veins of the abdominal parietes had become enlarged. The patient died afterwards of double pneumonia, and the diagnosis was confirmed by *post-mortem* examination. In the summer of 1859, I had under my observation a patient, who came to me from Karlsbad with all the symptoms of cirrhosis (ascites, slight jaundice, derangements of the gastric and intestinal digestion, small liver, large spleen, and anæmia). Under the use of Choleate of Soda and Infusion of Rhubarb, and small quantities of the mineral springs of Pyrmont (see Vol. I., p. 85, *Note.*—TRANSL.), the digestion improved, and the anæmia became less; after eight weeks, the ascites disappeared, whilst large venous cords might be seen upon the abdominal parietes, stretching upwards and downwards from the umbilicus.

[2] SAPPEY: Recherches sur un point d'anatomie pathologique relatif à l'histoire de la Cirrhose, *Bulletin de l'Académie de Médecine.* Paris, 1859, Tom. XXIV.

[3] The enlarged vein accompanying the ligamentum teres has hitherto been regarded as the umbilical vein, which has remained permanently open or again become pervious. Sappey maintains, that the observations which have been brought forward in support of this opinion are inaccurate, because the umbilical vein has no communication with the veins of the abdominal wall, and because it is improbable that the long, firm, fibrous cord of the obliterated vein should again become pervious.

larged epigastric and mammary veins being situated at a distance from the surface, and to the superficial subcutaneous veins being only implicated at an advanced stage of the disease, or often not at all. The circulation in these accessory branches of the portal vein, in the space between the abdominal wall and the liver, is reversed in cirrhosis; for, whilst the blood in these vessels formerly flowed towards the gland, in cirrhosis the portal blood passes through them to the abdominal veins, and is transmitted partly downwards into the epigastric vein, and partly upwards into the mammary vein. This afflux of blood to the veins of the abdominal walls gives rise to obstructions of the circulation, as a result of which, œdema of the feet and of the abdominal parietes sometimes make their appearance before there is any ascites. At a later period, owing to the operation of the same causes, phlebectases (*Phlebectasieen*) are formed beneath the skin of the abdomen, which pass outwards from the umbilical region, and, in most cases, present an elongated vascular tress-work (*Geflecht*) lying between the umbilicus and the epigastrium, and occasionally stretching downwards from the umbilicus towards the inguinal region. In many cases, this appearance of the veins is far from being an unimportant aid to diagnosis; but it must not be confounded with that dilatation of the veins extending over the entire abdomen, which is wont to occur in very extensive ascites, and especially when there is compression or obliteration of the vena cava inferior. In cases of the last-mentioned nature, it is usually accompanied by a varicose condition of the veins of the lower extremities.[1]

The collateral channels of circulation, which are developed in granular induration of the liver, rarely suffice to obviate the obstruction to the portal circulation and to compensate for the consequences of this obstruction. A series of anatomical lesions and functional derangements are gradually developed, which owe their origin to this cause. To these belongs, in the first place—

b. *The splenic tumor.* (*Der Milztumor.*)

Tumefaction of the spleen is by no means of such constant occurrence, as the operation of purely mechanical laws might have led us to expect, or as has been assumed by some observers. Out of 36 cases, I have found the spleen enlarged in 18, or in exactly one-half.[2] As a rule, the swelling

[1] When there is obliteration of the trunk of the portal vein, as now and then happens in cirrhosis, its accessory branches in the suspensory ligament can be of no assistance in carrying on the circulation, and then the blood is mainly transmitted by the anastomoses between the roots of the portal vein and the branches of the vena cava superior and inferior. We possess, however, few reliable observations in proof of this statement. Virchow (*Verhandl. der physik. medic. Gesellschaft zu Würzburg.* 1856. Bd. VII. s. 21) found a communication between the splenic vein and vena azygos, in a case of partial obliteration and ossification of the vena porta. The splenic vein presented three varicose sacs, which communicated with three varices of the vena azygos Reinaud (*Journ. hebd. de Méd.* 1829. Tom. IV. p. 137), in a case of partial obliteration of the vena portæ, observed greatly dilated veins beneath the capsule of the liver, which communicated with a dense network on the concave surface of the diaphragm; the phrenic vein also was in a varicose condition, as far as its junction with the subclavian.

[2] The ancient physicians were long ago aware, that the spleen frequently becomes enlarged in disease of the liver. Vesalius observed: "In morbis jecoris lienes magni frequentes." "Hepar scirrhosum; lien magnus." (Salzmann, in Boerhaave's *Prælectiones Acad.* Edid. Haller, Vol. III. p. 187.) Bianchi (*loc. cit.*, p. 159), indeed, attributed the sympathy between the two organs to the mechanism of the circulation. In more recent times, different opinions have been expressed, in reference to the frequency of enlargement of the spleen in cirrhosis of the liver. Whilst, on the one hand,

was of moderate extent; it rarely exceeded double or treble the normal volume; the average weight of the gland in 21 cases amounted to 0·24 kilogramme (8½ ounces avoird.), the largest weighed 0·88 kilog. (31 ounces av.), and the smallest 0·11 kilog. (3$\frac{8}{10}$ ounces av.). The organ was in most cases firm and dark-red; less frequently, its consistence was softer than natural. On three occasions, I have found the organ infiltrated with colloid matter; once there was an infarction, together with an atheromatous condition of the splenic artery; and, in four cases, the capsule was considerably thickened, and firmly adherent to the surrounding parts.

The absence of splenic enlargement is accounted for in several ways. In some cases the capsule of the organ is covered with fibrous patches, or calcareous plates, which offer great opposition to any distention; in other cases the swelling disappears, either because the obstructed blood gradually finds a free exit through the collateral channels, or because the tension of the portal vein has been reduced by profuse hæmorrhage from the vessels of the gastro-intestinal canal. Besides this, we must not forget, that the spleen contains an abundance of contractile tissue, and that, consequently, in addition to the pressure on the side of the obstructed blood, the contractility of the gland must be taken into account, the latter force counter-balancing the former, sometimes more, and sometimes less, completely. We are acquainted with the laws which regulate the activity of the muscular fibres of the spleen, except in so far that we know that poisonings of the blood, such as occur in typhus, pyæmia, intermittent fever, &c., produce a relaxation, from which a rapid enlargement results, even although there is no obstruction to the circulation. In countries where intermittent fever is of frequent occurrence, splenic enlargement appears to occur oftener in the course of cirrhosis, than it does at other places.

c. *Ascites.*

Ascites is a more constant accompaniment of granular induration of the liver, than enlargement of the spleen. I have met with it in 24 out of 36 cases, or in exactly two-thirds; in 7 of the cases the ascites constituted the sole form of dropsy throughout the entire duration of the disease, and in 17 cases œdema of the feet and other serous effusions were observable. The existence of abdominal dropsy depends, for the most part, upon the same causes as that of enlargement of the spleen. The more complete is the obstruction to the portal circulation, so much the more abundant is the effusion into the peritoneal cavity. The effusion usually commences at an early stage and goes on increasing for a long period, keeping pace with the advancing degeneration of the liver. At first it is easily overlooked, owing to the tympanitic condition of the intestinal canal; but, gradually, the fluctuation becomes more distinct, until at last the greatly distended abdomen assumes a barrel-shape, and the action of the diaphragm is more and more impaired. When paracentesis is performed in order to lessen the dyspnœa, the effusion returns after a

Bright, Smith, Oppolzer, and Bamberger have regarded it as an accompaniment of cirrhosis, which is only absent in exceptional cases: on the other hand, Andral, Budd, Monneret, and others, have usually failed to observe it. In 26 cases which came under Oppolzer's notice, splenic tumor was absent only in 4, and in 34 of Bamberger's cases it was absent only in 2. Bamberger is of opinion, that the swelling is only absent when there is some mechanical obstruction, marasmus or thickening of the capsule.

few days. In most cases the ascites precedes the œdema of the feet; but we must avoid regarding this circumstance as of too great value in diagnosis; cases not unfrequently occur, where, quite independently of any complication with disease of the heart, or kidneys, the dropsy makes its appearance at both places simultaneously.

The serous fluid is, as a rule, of a clear yellow character; in rare cases it is colored brownish or greenish from the admixture of bile-pigment, or reddish from the presence of blood; more frequently it is found to contain flaky coagula of fibrin, which are due to the supervention of an attack of general or circumscribed peritonitis. As regards its composition and specific gravity, I have been unable to detect any difference between it and other dropsical effusions into the abdominal cavity, such as occur in hydræmia, Bright's disease, diseases of the heart, &c., except that the effusion resulting from inflammation is always remarkable for the quantity of albumen which it contains.[1] A greater degree of hydrostatic pressure, such as must occur when the portal vein becomes obliterated, has produced no marked effect upon the density of the effusion in two of my cases; the solid contents amounted to 2·26 and 2·48 per cent., and the albumen to 1·06 and 1·04 per cent., in the two cases respectively. In some cases, sugar could be detected in the fluid; and in several, the fluid contained loosely-coagulated fibrin (or fibrin of retarded coagulation), and leucine.

The peritoneum is usually pale; we rarely find enlarged turgid veins in the mesentery, or on the serous surface of the bowel; in four cases only, have I met with extensive old ecchymoses of a black color. Traces of inflammation of the peritoneum, such as vascular injection and flaky exudations were of somewhat more common occurrence: these appearances were observed seven times (in the 36 cases), and in three only of the cases had the existence of inflammation been indicated during life by abdominal pain, slight fever, &c.; in three other cases, the process had remained latent. In one case (Observation No. XXXIV.), the peritonitis, without any other cause to account for it, supervened with such extraordinary rapidity, that death took place at the end of thirty-six hours, a large quantity of sero-purulent fluid having become effused into the peritoneal cavity. In one case only, was paracentesis the cause of peritonitis.

d. *The functions of the stomach and intestinal canal.*

As a rule, derangements of the stomach and intestines ensue, which are the more marked, the more the circulation in the blood in the roots of the portal vein is obstructed. The obstructed circulation manifests itself in the gastro-intestinal mucous membrane, by hyperæmia and abnormal secretion,—results which are particularly obvious in the stomach and large intestine, but are rarely noticed in the small intestine. Although the

[1] The quantity of solid constituents, in the fluid of ascites resulting from cirrhosis, varied in six analyses from 2·04 to 2·48 per cent.; that of the albumen from 1·01 to 1·20. In hydræmia and Bright's disease, the solid contents amounted to from 2·04 to 2·8 per cent., and the albumen varied from 1·01 to 1·2 per cent.; in diseases of the heart, the former was 1·76 per cent. and the latter 1·18 per cent. In cirrhosis complicated with slight peritonitis, the quantity of the solid contents rose to 3·59 per cent., with 2·6 per cent. of albumen; in tubercular peritonitis it amounted to 5·2 per cent. with 4·2 of albumen; and in simple chronic peritonitis it was 5·5 per cent., with 3·86 per cent. of albumen.

obstruction is uniform throughout the entire portal system, its effects, just like those of mechanical hyperæmia of the liver in consequence of heart disease, are far from being uniformly distributed; sometimes they are more marked at one place, and sometimes at another, according as the mode of distribution of the capillaries, the contractile force of the muscular tissue (as in the case of the spleen), and other partly unknown influences are favorable or the reverse. In many cases, the exalted pressure of the blood leads to rupture of the vessels and hæmorrhage, which usually takes place from the mucous surface; more rarely the blood is infiltrated into the tissues of the bowel, and produces erosions, which end in the formation of ulcers; bloody suffusions of the serous coat are occasionally observed; it is only in exceptional cases that varices filled with coagulated blood are found in the mucous membrane. Of the 36 observations, the mucous membrane of the stomach was pale in 8, and in 26 it was in a state of catarrhal tumefaction, and of a more or less deep livid hue. Hæmorrhagic erosions existed in 4 cases, and cicatrices in 2; in 6 cases, the stomach and intestine contained a bloody fluid. In the large intestine, these changes were much less frequently observed. In 13 cases only was the mucous membrane of this portion of the bowel softened and of a livid hue; in 5 cases, there were superficial catarrhal ulcerations. The small intestine was rarely implicated, and never more than very slightly. Thus, on the whole, the consequences of the obstruction to the circulation, manifest themselves more frequently in the stomach and intestines than in the spleen, the exemption of this organ being apparently due to its powerful muscular tissue. The ascites coincides in frequency with the stomach affection.

From what has just been stated it follows, that derangements of the gastric and intestinal digestion are the ordinary accompaniments of granular induration of the liver. These derangements contribute greatly to the induction of the cachectic state, which is rarely absent in the advanced stage of the disease. The more the digestion is impaired, the sooner does the nutrition suffer, and the sooner does the patient lose flesh and strength. In addition to the disordered digestion, another important element in the production of cachexia, is the impaired absorption of the digested materials on the part of the branches of the portal vein, which must necessarily result from their over-distended condition. The more the pressure of the blood in these vessels is increased, the greater is the impediment offered to the absorption of materials from the stomach and intestine in order to become incorporated with the blood. The importance of this last abnormal condition will be estimated, according to the view which is entertained concerning venous and lacteal absorption. In my opinion, the truth is by no means established, of what most German physiologists assume in opposition to the French, viz.:—that the digested albuminous substances are only absorbed by the lacteals, while to the veins is assigned the absorption of the water, salts, sugar, &c. The endosmotic properties of peptone have not yet been sufficiently examined, and the conclusions which are drawn from the nature of albuminous substances, must not be extended until this is done. But, whatever view is adopted in reference to this question, there can be no doubt that, with the obstruction to venous absorption, a powerful agent in the processes of digestion is impaired. In this way, the fact is accounted for, that many patients become cachectic, although their appetite does not fall off, and although their impaired nutrition cannot be referred to diarrhœa, excessive ascites, &c.

The symptoms referrible to the stomach, during the progress of cir-

rhosis, are very various. Cases occur where the appetite is not at all impaired; this I have observed in 7 out of 36 cases. Not at all unfrequently, the disease commences with symptoms of intense gastric catarrh, painful tension of the epigastrium and hepatic region, nausea, vomiting, loaded tongue, jaundiced tint of the countenance, constipation, &c. These symptoms usually disappear after some time, to return at a later period, often not until after the lapse of months, and then to remain persistent. In other cases, the symptoms of disordered gastric digestion do not make their appearance until a later period, after the first indications of ascites have become obvious; they then gradually increase in intensity. At first, the patient complains of uneasiness after a copious meal, or after taking food to which he has not been accustomed; after a time, even light articles of diet are not tolerated, without producing a feeling of tightness at the epigastrium, heartburn, &c. These symptoms are either persistent, or intermit from time to time, until some new cause calls them forth again. In the cases where hæmorrhagic erosions or ulcers are formed, the tenderness at the epigastrium becomes intense, the vomiting is constant, and not unfrequently blood is ejected from the stomach in large quantity. Hæmatemesis, however, may also occur without any loss of substance in the mucous membrane, owing merely to rupture of the capillary vessels in consequence of the obstruction to the circulation.

The bowels are in most cases costive, particularly in the early stages of the disease; at a later period, diarrhœa is more frequent. The gastric digestion is almost invariably accompanied by flatulence, which is the more troublesome, the more the abdominal cavity has already been filled up by ascites. The diminished secretion of bile and the weakened contractility of the muscular fibres of the intestine are the proximate causes of the flatulence. The stools vary in character: in the early stage of the disease they are usually normal, except that they are drier, and often covered with a thick layer of vitreous-looking mucus; at a later period, they usually assume a paler color, which is the more marked, the more that the formation of bile is diminished in consequence of the advancing destruction of the gland. We then observe sometimes, what Graves has already called attention to, one portion of the fæces pale and clay-colored, and another darker, according as the sparingly and rarely-filled gall-bladder empties its contents into the bowel or not. As the disease progresses, diarrhœa frequently supervenes; the stools are of a pale grayish-yellow color; occasionally they are mixed with blood or with flakes of exudation, and in exceptional cases they assume the appearance of rice water, or of water in which flesh has been macerated (*fleisch-wasserartig*). I have met with persistent diarrhœa ten times in 36 cases, but in two of the ten cases the cirrhosis was complicated with tubercular ulceration of the intestine. The diarrhœa becomes more urgent shortly before the fatal termination.[1] Profuse exhausting hæmorrhage from the intestinal canal is rare in cirrhosis. Enlargements of the hæmorrhoidal veins are also by no means frequent; their occurrence has been assumed by many writers *à priori*, rather than actually observed.

c. *Alterations of nutrition and other consecutive disorders.*

The nutrition of patients laboring under cirrhosis suffers at an early period. They emaciate; the fatty cellular tissue disappears from their

[1] The *diarrhœa hepatica* of the ancients. Bianchi thought it proceeded "a transcursu lympharum per hepar impedito." (*loc. cit.*, Vol. I. p. 109).

muscles, while at the same time they lose strength. It is only on rare occasions, that we meet with a patient suffering from this disease, whose appearance and strength do not indicate the existence of a deep-seated cachexia. The color of the skin becomes pale and earthy; often it resembles the whiteness of anæmic persons, whilst in other cases it presents a yellowish tint, which may pass through many intermediate grades into true jaundice. This yellow tint I have met with seven times (in 36 cases), but in two only was it intense ; in most cases the shade was a dirty grayish-yellow, distinctly marked over the upper half of the body, but gradually disappearing below.

The jaundice of cirrhosis, when it attains a high grade, depends upon catarrh of the bile ducts, or upon compression of the hepatic duct by enlarged lymphatic glands in the fissure of the liver (Observation No. XXXIV.), or upon some other complication. The slighter forms, which are more common, are accounted for by the injuries usually inflicted on the minute biliary ducts, by the newly-formed connective tissue in the circumference of the hepatic lobules. They are due to the same cause as the jaundiced condition of the liver itself, to which the cirrhosis is indebted for its name.

The emaciation and debility of patients laboring under cirrhosis are the result of the injuries, usually sustained by the process of assimilation. In the first place, there is an enfeebled condition of the gastric and intestinal digestion ; the appetite fails, and there is a defective elaboration of the ingesta not merely in the liver, but, owing to the scanty secretion of bile, in the intestine also. This, however, is not the only cause ; we meet with patients falling off, even when the appetite remains unaffected, and there is no symptom of disordered digestion present. A second cause, which is the more important the greater the obstruction to the circulation through the liver, is the diminished absorption by the roots of the portal vein. In addition to this, we must blame the impaired action of the hepatic tissue upon the metamorphosis of matter, and the injurious effects of the dropsy upon the composition of the blood.

No important abnormal conditions of the organs of circulation are met with during the progress of cirrhosis ; in isolated cases the affection is accompanied by a febrile excitement of the heart's action, intercurrent attacks of which are, also, now and then observed, going along with severe exacerbations of the gastro-enteric catarrh, peri-hepatitis, &c. ; but, on the whole, this is a rare symptom. Most cirrhotic patients die without the pulse undergoing any essential alteration, except an increased *frequency* and *smallness* attendant upon the anæmia. As might have been expected, the pulse becomes slower on the supervention of jaundice in this as in other affections. (See Vol. I., p. 77.)

Capillary hæmorrhages are not very uncommon in cirrhosis; their production cannot be accounted for more easily in this disease, than in acute atrophy of the liver and in other forms of typhoid jaundice. They occur most frequently in the stomach and intestinal canal, where there is also a mechanical cause in operation, but they are also frequently observed at places where such a cause cannot act in any way, as in the form of petechiæ upon the skin, hæmorrhages from the mucous membrane of the nose and mouth, hæmorrhagic effusions into the serous cavities, hæmorrhages of the cerebral membranes, pulmonary apoplexies, &c. They are particularly frequent in cases which terminate fatally under symptoms of acholia, but they are also met with independently of the existence of any severe nervous symptoms. Bright long ago expressed the opinion, that they

were due to an altered composition of the blood; but as yet no one has positively proved this. The remarks already made on the hæmorrhages, which occur in acute atrophy of the liver, are equally applicable here.

In the later stages of the disease, the respiration is usually much impeded, owing to the ascites and meteorism, and in most cases the dyspnœa is so great as to necessitate the repeated performance of paracentesis.

Pneumonia, pleuritic exudations, and pulmonary œdema, with the respiratory symptoms pertaining to these affections, not unfrequently precede the fatal termination; pneumonia is particularly common in the cirrhosis of drunkards. I have observed it in 4 (of 36) cases.

The renal secretion usually diminishes in quantity; the urine becomes scanty, and red or brown-colored, and often deposits a sediment of a red or bluish-red hue; in rare cases, it is pale and ammoniacal. When the cirrhosis is accompanied by jaundice, the urine is more or less deeply tinged with bile-pigment. In not a few cases, it is albuminous, owing to the disease of the liver being complicated with degeneration of the kidneys. I have observed this eight times in 36 cases.

Sometimes the secretion of urine in cirrhosis is reduced to a very small quantity, especially when, after the performance of paracentesis, the ascitic fluid continues to flow off freely; in one case, six ounces only were passed in 24 hours. (See cases subsequently detailed.) We might have expected to have found the destruction of the hepatic parenchyma, accompanied by alterations in the quality as well as in the quantity of the urine, but I have not succeeded in detecting any peculiar products in this secretion. Leucine has been repeatedly sought for, but I have never succeeded in discovering it.

The functions of the nervous system, as a rule, remain undisturbed; the patients endure their sufferings in a gloomy or collected frame of mind, according to their natural disposition, and commonly retain their consciousness until the last. In rare cases, however, we see severe nervous symptoms,—delirium, convulsions, and coma, just as the same symptoms occur in acholia resulting from acute atrophy of the liver. Examples of this nature have already been detailed and commented on in the first volume (p. 170).

5. *Complications.*

Besides the disease of the liver, we very commonly meet with affections of other organs, which are sometimes independent of the hepatic affections, but, at other times, are nearly or remotely connected with it. To the former class belong tubercle and emphysema of the lungs (the former of which I have observed six times, and the latter thrice, in 36 cases), and also diseases of the heart, carcinoma, &c. Other complications originate from the same cause as the cirrhosis, such as the morbus Brightii of drunkards, delirium tremens, lardaceous spleen and kidneys, syphilitic affections of the bones, &c. Lastly, others supervene as the direct or indirect consequences of the degeneration of the liver, such as secondary pneumonia, pleurisy, peritonitis, dysentery, various hæmorrhages, and the remaining consecutive morbid processes already mentioned.

It is obvious that the clinical characters of cirrhosis may be greatly modified in this way; whilst the symptoms, to which the complications give rise, being superadded to those of the cirrhosis, may often obscure them to such an extent, as to render the diagnosis very difficult.

6. *Duration and Progress.*

It is usually a difficult matter to determine the date of commencement of induration of the liver; this can only be done with any degree of certainty, when the affection commences with well-marked hepatitis. As this rarely happens, the calculation of the time is in most cases uncertain. It is not sufficient to date from the commencement of the ascites, or of the symptoms of indigestion, inasmuch as both do not usually supervene until after the hepatic disease has lasted for a long time. It is generally agreed that the disease invariably runs a chronic course, and not unfrequently lasts upwards of a year, or even extends over several years. Cases do occur which terminate fatally so early as at the end of one or two months; but in this case, complications, such as cardiac diseases, pulmonary emphysema, &c., usually assist in hastening the fatal termination.

No real intermission can be recognized in the progress of the disease. It either advances steadily, or makes occasional rapid strides when fresh exciting causes come into play.

7. *Modes of Termination.*

After the disease is fully developed, the termination is always unfavorable. The complete destruction of the glandular substance cannot be repaired in any way; and it is only under favorable circumstances, that the collateral channels compensate for the obstructed circulation through the portal vein. It is possible, yea probable, that by this last means the functional derangements compromising life may be relieved, at all events, in the slighter forms of hepatic degeneration, and that thus a cure, though an imperfect one, may ensue. At all events, this opinion is favored by the observations which are now and then made at *post-mortem* examinations, where cirrhosis is found unexpectedly in individuals who have died of some other disease, and who have not presented during life any of the disorders which usually result from cirrhosis. (See Observation No. XXV., and others.) It is further countenanced by the disappearance of the dropsy, which is occasionally observed to take place during life, after the abdominal veins become enlarged; such cases, however, are entirely exceptional, and do not at all subvert the general rule. It is far more possible to arrest by appropriate treatment the chronic hepatitis, which in some cases announces the commencement of cirrhotic degeneration; but this is a prevention of the cirrhosis rather than a cure. It is not an easy matter to determine how often the disease is thus prevented, because the primary inflammatory process rarely exhibits such characteristic symptoms, as to enable us to foresee with certainty the danger of cirrhosis supervening.

Thus, in by far the majority of cases, the disease terminates fatally. In most cases, the immediate cause of death is the exhaustion induced by the impaired nutrition, and by the deficient formation of blood; and then the disease advances slowly and gradually towards its fatal termination under symptoms of marasmus and general dropsy. In other cases, death results from the supervention of acute diseases, such as febrile gastro-enteric catarrh, peritonitis (Observation No. XXXIV.), pneumonia (Observation No. XXIX.), pleuritic exudations, œdema of the lungs, hæmorrhage from the bowels, dysentery, gangrenous erysipelas of the œdematous skin, &c. Most frequently, it is catarrh of the stomach and intes-

tines which puts an end to life. The patient suddenly loses all appetite for food; the tongue is covered with a thick brown coating, which soon becomes dry; diarrhœa sets in, with pale mucous stools, sometimes tinged with blood, and often attended by vomiting; whilst, at the same time, the pulse rises to 110 or 130, and, after a few days, typhoid somnolence and collapse supervene.

In a few cases only does death ensue under the ordinary symptoms of acholia, which have already been described in detail. I have met with this mode of termination only three times in 36 cases. (See Observations No. XX. and XXI. in Vol. I. pp. 170 and 171.)

8. *Prognosis.*

From the statements just made it is obvious, that the prognosis must always be in a high degree unfavorable; death, as a rule, is inevitable. The only question, therefore, is the possibility of relieving those disorders which mainly threaten life, and of delaying the fatal termination. Our success in accomplishing these objects depends upon various conditions. In the first place, there is the stage of the disease, for in an advanced stage of degeneration all treatment is impotent. A second condition is the existence or absence of complications; heart diseases, simple or lardaceous degeneration of the kidneys, and lardaceous spleen, have a powerful effect in accelerating the progress of the disease. Besides these conditions, there are the external circumstances of the patient, his ability to obtain careful nursing and attention, &c.

9. *Diagnosis.*

In practice, it is by no means always an easy matter to diagnose cirrhosis of the liver with certainty, especially when, as is usually the case, we have only an opportunity of observing the disease at one stage of its progress. The facts upon which our diagnosis must rest, are the following:—persistent derangement of the digestion with no obvious organic disease of the stomach, ascites, tumefaction of the spleen, diminution of the liver, increasing paleness of the fæces, cachexia. These indications render the presence of cirrhosis probable, but not certain, for the same train of disorders may proceed from simple chronic atrophy of the liver, from pylophlebitis adhæsiva, from compression of the portal vein by tumors or bands of areolar tissue in the fissure of the liver, from chronic inflammation of the hepatic veins, and, in short, from all those numerous morbid processes, which give rise to gradual obstruction of the circulation in the portal vein, and reduce the volume of the gland. A correct diagnosis is only possible, when we can succeed in distinguishing by means of palpation, those alterations in the form and consistence of the gland which characterize granular induration. Besides this, it is important to note the mode of development of the symptoms of obstruction, which in disease of the hepatic vessels is usually much more rapid than in cirrhosis,[1] and also the predisposing causes to which the patient has been exposed before the attack, such as the abuse of spirituous liquors, &c.

[1] In two cases, subsequently detailed, closure of the hepatic vessels was diagnosed during life, by this means alone.

From this it appears, that in cases where extensive ascites or other causes prevent the liver from being felt, the diagnosis must often continue to be nothing more than probable, and that simple atrophy of the gland in particular cannot, in many cases, be distinguished from cirrhosis.

From diseases of the liver attended by enlargement of the organ, such as carcinoma, echinococci and colloid (amyloid) infiltration, the diagnosis is in general easy, because the ascites and other signs of obstructed circulation are absent in these affections, and because an increase in the volume of the gland is only met with in cirrhosis as a temporary phenomenon at the commencement of the disease, and even then not invariably. The gland is only permanently enlarged when, in addition to the granular degeneration, colloid infiltration or extensive deposition of fat is present; in such cases, we cannot fail in discovering the nodulated surface. But, as frequently happens, especially in syphilitic patients, cicatricial contractions may coexist with these enlarged forms, and, consequently, large nodules may be developed on the surface, of such a character as to render the diagnosis from cancer extremely difficult. Cases illustrative of this difficulty shall be detailed farther on.

Cirrhosis may also be confounded with ascites resulting from chronic peritonitis, whether this be simple, tubercular or cancerous, and the more readily, as the size of the liver is, in most cases of chronic peritonitis, apparently diminished in consequence of its displacement. The diagnosis here must mainly depend upon the greater tenderness of the abdomen in peritonitis,[1] and upon the local and general symptoms characteristic of tubercle or cancer; the splenic tumor, moreover, is usually absent, and the gastro-enteric catarrh less constant in peritonitis. The diagnosis is greatly simplified by the direct examination of the liver, after the performance of paracentesis.

10. *Varieties of Granular Induration of the liver, and Illustrative Cases.*

Although the main symptoms of cirrhosis of the liver are always the same, its clinical history presents manifold varieties, according to the mode of origin of the morbid process, and the existence of complications, which are for the most part dependent upon the primary causes of the disease. The simplest form is that observed in drunkards; here the hepatic affection either remains uncomplicated, or is associated with Bright's disease, or sometimes with pneumonia, delirium tremens, &c. The disorders of the digestive organs are as a rule very prominent, because in addition to the obstruction of the circulation, the mucous membrane of the stomach is kept in a state of constant irritation by the imbibition of spirituous liquors.

The cirrhosis which occurs in syphilitic patients, is often accompanied by amyloid degeneration of the spleen and kidneys, and sometimes of the liver and mucous membrane of the intestine. The cachexia attains a high grade at an early period. In addition to this, the remains of syphilitic inflammation are found in the liver; the gland is divided into lobes by bands of areolar tissue penetrating more or less deeply into its substance, whilst the cirrhotic induration is restricted to isolated masses.

[1] Experience, however, has taught me, that we must not always reckon upon the presence of this symptom in peritonitis.

The cirrhosis, which is developed in the course of intermittent fever, is usually accompanied by enlarged pigment-spleen.

In cases were chronic inflammation, originating in the capsule or in the diaphragm, attacks the glandular substance, I have observed the portal vein or the hepatic veins implicated to a great extent, the glandular parenchyma at different places uniformly indurated, and the outer surface lobulated.

The following observations will elucidate more fully many details :—

Observation No. XXIII.

Extensive Ascites without Œdema of the Feet.—Disordered gastric and intestinal Digestion.—Urgent Dyspnœa—No obvious cause for the disease.—Temporary improvement.—Increase of the Dropsy.—Administration of purgatives.—Paracentesis.—Death.

Autopsy.—Cirrhosis of the Liver.—Thickening of the walls of the Vena Portæ.—Splenic Tumor.—Fatty Degeneration of the Muscular Tissue of the Heart.—Sugar and Leucine in the ascitic fluid.

Georges, a corn-broker, aged 66, sought relief at All Saints' Hospital, on December 5th, 1856, having suffered for five weeks from ascites without any œdema of the feet. The thoracic organs presented no important morbid change; elevation of the diaphragm and catarrhal râles at the lower and back part of both lungs were the only causes for the dyspnœa, which could be detected. The abdomen was of a vaulted globular form, and presented distinct fluctuation; the abdominal veins were much enlarged. The hepatic dulness in the mammary line amounted to 3 centimètres (1⅛ English inch); the spleen was considerably enlarged and projected beyond the margins of the eleventh and twelfth ribs. Appetite slight; tongue clean; one pale, thin stool daily; a considerable amount of tympanites; urine scanty and red. The patient stated, that previously he had always enjoyed good health, and that he had never been addicted to the abuse of spirituous liquors.

Infusion of Rhubarb with Liquor Ammoniaci Anisatus,[1] and a light animal diet were prescribed.

The difficulty of breathing diminished, the appetite improved, and by the end of ten days the patient felt himself considerably relieved, when the ascites and tympanites again increased, and the dyspnœa became intense. The use of Decoction of Colocynth brought away copious watery evacuations from the bowels, but was followed by no improvement. Recourse was therefore had to paracentesis, and about ten quarts of clear serous fluid were drawn off. The operation was succeeded by a temporary abatement of the alarming dyspnœa; but, a few days after, fatal collapse ensued. Immediately after the tapping, the hepatic dulness in the mammary line was ascertained to measure 6 centimètres (2·36 Eng. inches).

Autopsy.

Nothing abnormal in the cranial cavity.

The mucous membrane of the bronchi was injected, and at some places

[1] Composed of anise oil 1 part, liquor ammoniæ fortis 8 parts, and rectified spirit 32 parts.—Transl.

ecchymosed; the lungs were congested and œdematous. The muscular tissue of the heart was pale, friable, and fatty, particularly the left ventricle; the valves were normal. The spleen was enlarged by about one-half; its capsule was opaque and thickened, and the pulp unusually soft and congested. The abdominal walls and omentum loaded with fat; the mucous membrane of the stomach and small intestine pale, that of the colon thickened and of a livid hue. Fæces solid and brown. Kidneys normal, with the exception of a few cysts of the size of a pea, and calcareous infarctions of the pyramids.

The liver was adherent, at many places, to the surrounding parts. It was somewhat reduced in size, and covered with nodules from the size of a linseed to that of a pea; on section, larger or smaller groups of similar nodules were observed, some of them of a deep jaundiced hue. The walls of the larger branches of the portal vein were thickened, and in whiteness and firmness resembled the arteries; they contained tarry, thick fluid blood, without any large clots. The secreting cells of the granulations, which were surrounded by thick bands of areolar tissue, were loaded partly with fat and partly with pigment.

A small quantity of turbid grayish-yellow fluid was found in the gall-bladder.

The fluid drawn off from the abdominal cavity by tapping abounded in sugar, and also contained leucine.

Observation No. XXIV.

Disordered gastric Digestion.—Vomiting.—Diarrhœa.—Ascites.—Œdema of the Feet.—Puncture of the Abdomen.—Splenic Tumor.—Liver small, with nodulated surface.—Death.

Autopsy.—Cirrhotic and lobulated Liver.—Thickening of Glisson's Capsule.—Firm adhesion of the lower surface of the Liver to the adjoining parts, and also of the indurated Pancreas to the Vertebral Column and Retro-peritoneal Glands.—Recent Peritonitis.

Susanne Springer, a female day-laborer, aged 54, was admitted on July 30th, 1852. Up to three years before, the patient had enjoyed good health, and menstruated regularly; but, ever since, she had been in a sickly state. Her symptoms were pains in the upper part of the abdomen, particularly after eating, failing appetite, and constipation; while the abdomen became gradually enlarged to a considerable extent. In May, June and July of 1852, hæmorrhages took place from the sexual organs, which in July became so copious, that the patient applied for medical relief. With these hæmorrhages the swelling of the abdomen was reduced; but the patient was attacked with diarrhœa, and vomiting of a greenish-bitter substance, which persisted for a long period and greatly exhausted her. Eight days before admission, the vomiting and diarrhœa had both subsided. A fortnight before admission, the lower extremities became œdematous to above the knees; the ascites increased greatly; the respiration was impeded; the cutaneous veins upon the abdomen and chest became distended; and the urine was diminished in quantity, but contained no albumen. The upper part of the body was much emaciated. There was dulness over the lower third of the left side of the thorax, while above, a rough expiratory murmur was audible. There was no dulness in the epigastrium; owing to the

anasarca, the boundaries of the spleen and of the right lobe of the liver could not be defined. Under the use of diuretics and bitter remedies, the quantity of urine increased and the appetite improved; but the ascites increased. On the 4th of August, the patient was ordered Infusion of Rhubarb with Spirit of Nitric Ether. This was followed by a firm clay-like stool, while the œdema of the feet diminished. On the 5th, paracentesis was performed; 12 quarts of clear, opalescent, highly albuminous fluid were drawn off. On the 8th, the abdomen was painful when touched, and by the 11th the ascites had increased to its former amount. Infusion of Rhubarb with Bitartrate of Potash was prescribed. On the 23d, there had been an increase in the amount of urine for some days. On September 1st, paracentesis was repeated; after the fluid was drawn off, the liver could be felt along the lower margin of the right ribs, with its margin sharp and covered with nodules. After this the patient had from two to four thin, pale stools daily. Decoction of Cascarilla Bark with Tincture of Nux Vomica was prescribed, without any benefit; the diarrhœa increased, and the patient lost strength. The urine was of normal quantity and color, and free from albumen. Death from exhaustion occurred on the 24th September.

Autopsy on September 26th.

Serous effusions in both pleural sacs, but most abundant in the left; the lungs emphysematous at their anterior margins, at other places œdematous; a pulpy calcareous deposit the size of a cherry-stone, surrounded by gray indurated tissue, in either apex.

The pericardium and heart normal; the large flap of the mitral valve thickened but not shortened; the blood in the right side of the heart fluid, in the left, coagulated in clots.

The mucous membrane of the stomach near the pylorus was of a deep slaty-gray hue, but not thickened. There were patches of vascular injection at many places near the lower extremity of the small intestine; the cæcum and the large intestine, throughout its entire extent, was of a slaty color, the mucous membrane being slightly œdematous and the solitary glands enlarged. The contents of the bowel were grayish-yellow and pultaceous. The mesenteric glands contained pigment, and were hard and flattened. The lymphatic glands surrounding the large vessels of the pelvis and along the vena cava were enlarged, and on section exhibited the lustre of lardaceous deposit. The areolar tissue lying along the vertebral column was increased, and of a dense character, particularly in the region of the pancreas, which was almost immovably adherent to the vertebral column, and which appeared firmer and more finely granular than in the normal state. The increase of the areolar tissue extended to the *porta hepatis;* at this place, portions of the great omentum, the under surface of the liver, the duodenum, the pyloric end of the stomach and the right curvature of the colon were all drawn closely towards one another, and firmly adherent. The coats of the gall-bladder were thickened; its cavity would barely hold a pigeon's egg, and its contents consisted of a grayish-white mucus; its mucous surface was of a slaty-gray hue; the ductus hepaticus was much enlarged, and of a bright-yellow color. The liver was divided by deep fissures into large lobes, and exhibited throughout granulations the size of a pea, which, on section, appeared dry and grayish-yellow. The organ was reduced in volume, but not more than about one-third. On the cut surface, the divided extremities of the branches of the

portal vein and of the bile ducts were imbedded in thick white layers of dense areolar tissue.

The kidneys were of normal size; their capsule was easily separable. Their outer surface was granular, and also exhibited deep and superficial cicatrix-like depressions. The cortical substance was much shrivelled; the parenchyma was firm and tenacious.

The spleen was five inches long and three inches broad; it was dark-brown and contained but little blood. The uterus and ovaries were atrophied. The peritoneal cavity contained a quantity of turbid-yellow fluid; there were fibrinous deposits in the cavity of the pelvis, and upon the abdominal viscera the peritoneum itself was vividly injected, opaque and dry. The wounds of both punctures were completely cicatrized, and the corresponding part of the peritoneum was marked by a halo of gray pigment three or four lines in diameter.

In this case, as would appear from the patient's history before admission, with which the result tallied, the disease commenced as chronic peritonitis, which extended along the retro-peritoneal areolar tissue, the pancreas, stomach, and lesser omentum, as far as Glisson's capsule in the *fossa hepatis*, and penetrated, with this, deep into the substance of the liver. This peritonitis accounted for the numerous adhesions of the organ, as well as for its lobulated character. The first effusion into the peritoneal cavity, which partially disappeared after the occurrence of the uterine hæmorrhage, also dated from this peritonitis.

The case illustrates the statements made above as to the consequences of peri-hepatitis. (See likewise Observation No. XXXV., and particularly No. XXXVII.)

Observation No. XXV.

Paralysis (Lähmung) of the Hypoglossal and Facial Nerves, and incomplete paralysis (Parese) of the muscles of the trunk and extremities.—Dysentery.—General convulsions.—Death.

Enlargement and abnormal mobility of odontoid process of second vertebra.—Granular Induration of Liver without any obvious cause.—Splenic Tumor.—Slight Ascites.—Dysenteric Inflammation of the large Intestine.

Carl Zeppner, a peasant's son, aged 10, was admitted on June 1st, 1854.

Up to a year before admission, this boy had enjoyed good health, and in bodily and mental development had not been behind other children of his age. After bathing on one occasion, when he had been violently plunged by his companions with his head under the water, and kept there for some time, he complained of headaches and lassitude, became oblivious, began to stammer and to speak slowly and with difficulty, and gradually lost the power over his extremities. He often kept his bed for days on account of weakness; and, when he wished to use his limbs, they were thrown into violent tremors. In other respects, his intelligence was not impaired and his sensibility not diminished. Since Christmas, he had completely lost all power of speech, and had been unable to swallow any solid food. His parents had not noticed that he had been affected with convulsions, vomiting, or constipation, and his appetite had throughout been moderate.

On admission, the boy appeared somewhat emaciated, but in other respects there was nothing else of an abnormal character to be observed in his external appearance. He was unable to stand or sit upright, or to hold anything in his hands; all movements were possible, but were performed very slowly and awkwardly; the sensibility was unaffected. His countenance exhibited an air of indifference and failed to express either pain or pleasure; when the boy was told to distort his features, scarcely perceptible movements of the muscles of the face ensued, and the eyeball was moved instead. The mouth opened slowly and incompletely. The tongue, which appeared somewhat flattened, but which was in other respects normal, was seized with a feeble tremor, when the patient was ordered to move, or protrude it, or to speak; the prick of a needle, however, could be felt in both halves of the organ; cold and warmth, and both sour and sweet things, could be distinctly appreciated. The patient could swallow nothing but liquids, and even these with difficulty. The hearing and sight were unimpaired.

For some days, the patient had been troubled with a cough, but nothing abnormal could be detected in the lungs. The sounds of the heart were normal; pulse 90. The appetite was moderate; the bowels somewhat confined; no involuntary evacuation. The urine was dense, but free from sugar. The abdomen was free from pain, and slightly tympanitic.

From time to time the difficulty of deglutition increased, and at the same time the movements of the extremities were imperfectly performed.

On the 13th June, without any obvious cause, the patient was attacked with profuse diarrhœa and painful enlargement of the abdomen. Gradually the stools assumed a dysenteric character, the temperature of the body rose, and the pulse reached 116. Was ordered Decoction of Calumba and the Watery Extract of Nux Vomica.

From the 16th, the stools and urine were passed involuntarily. During the night of the 18th, the patient completely lost his consciousness and ground his teeth; the head, which previously had been invariably turned backwards in the bed, was kept perseveringly directed towards the right side; the countenance was much flushed; diarrhœa moderate; pulse 124. Was ordered leeches and an ice cap to the head. On the afternoon of the 18th, violent general convulsions set in; the pupils were dilated, and the respiration impeded. After lasting for four hours, the convulsions terminated in death.

Autopsy, 13 *hours after death.*

The body was emaciated, but there was no œdema. The muscular tissue was atrophied, but not degenerated.

The form of the skull was normal. The cerebral membranes were much injected, and the veins distended with dark blood. The substance of the brain was soft, particularly in the fornix and corpus callosum; everywhere it contained much blood. There was nothing abnormal in the lateral ventricles; but, beneath the lining membrane of the fourth ventricle, were extravasations of blood, the size of a linseed. In other respects nothing abnormal could be found in the substance of the brain, or at the base, or at the origin of the nerves.

The medulla oblongata was firmer and more tenacious than in the normal state. The odontoid process of the second vertebra was unusually

prominent, and this prominence was increased when the base of the cranium was moved laterally or forwards, which could be done with remarkable facility; the ligamentous apparatus of this vertebra appeared considerably relaxed, but, in other respects, was unaltered.

The tip of the tongue was remarkably thin; the pharynx and œsophagus normal. The larynx and trachea contained bloody mucus; and their lining membrane was injected. There was no important change visible in the pleuræ or lungs, with the exception of pulmonary hypostasis. Heart normal.

The peritoneal sac contained a small quantity of serum. The mucous membrane of the stomach was covered with bloody serum, and in the neighborhood of the pylorus was relaxed and livid. The mesenteric glands were enlarged, and of a melanotic hue. The serous coat of the small intestine exhibited patches of lividity; its mucous membrane was tumid. The mucous membrane of the ascending colon was of a bluish-black tint, and here and there covered with flakes of exudation; this color became fainter in transverse colon, and disappeared entirely towards the sigmoid flexure. The kidneys and urinary bladder were normal. The spleen was enlarged—6½ inches long, 4½ broad, and 1 inch thick—weight 0·36 kilogramme (12·69 ounces avoird.); its capsule was thickened and opaque; in its parenchyma, numerous extravasations of blood could be seen passing inwards from the surface in the form of wedges.

The liver was small, and its convex surface was adherent by numerous bands to the diaphragm. Its surface was covered with nodules, varying in size from a pea to a bean, and similar formations could be seen in its interior, where they were separated from one another by broad bands of areolar tissue. The consistence of the organ was tenacious and leathery. The gall-bladder contained only a small quantity of pale bile.

The three following cases of cirrhosis of the liver are worthy of notice, inasmuch, as they were preceded by obstinate intermittent fevers. In one of them (No. XXVII.), not one of the causes existed which are wont to give rise to cirrhosis; in the two others, the abuse of spirituous liquors, &c., could not with certainty be excluded.

Observation No. XXVI.

Persistent Intermittent Fever.—Irregular habits of life.—Gastric Catarrh.—Slight Jaundice.—Cachexia.—Ascites.—Paracentesis.—Collapse.—Death.

Autopsy.—Finely-granular Cirrhosis of Liver.—Splenic Tumor with slight pigment-deposit.—Catarrhal Tumefaction of the Mucous Membrane of the Stomach.—Cicatrices in the Duodenum.—Typhus (sic) Cicatrices in the Ileum.

Rosina Tietze, aged 28, was a patient in the clinical department of All Saints' Hospital, from the 14th of April to the 20th of May, 1857.

Up to the beginning of 1857, she had been at service in the country, working in the morning in the house, and in the afternoon out of doors, and she had then been in the habit of drinking from one to two glasses of brandy daily. During the previous summer and autumn, she had suffered, first for six weeks, and afterwards for four, from a tertian inter-

mittent fever, for which she had been treated in the Ohlau[1] Infirmary. During January and February, she seemed to have led a very irregular course of life. The patient stated, that for four weeks before admission her appetite had failed, and she had been troubled with a feeling of fulness in the right hypochondrium and epigastrium, and during the same period she had observed a swelling of the abdomen, and a pale-yellow tinge of the skin. Three weeks before, her feet had become swollen.

The patient was jaundiced and remarkably emaciated; the skin was everywhere dry, fissured, and covered with branny scales; there was extensive ascites and likewise slight enlargement of the veins upon the abdomen. The lower extremities were moderately œdematous; the upper were free from œdema.

Percussion of the chest presented nothing abnormal; loud sibilant and moist râles were audible over the back part of both lungs; the patient complained of a troublesome dry cough. Heart normal.

The hepatic dulness was completely absent at the epigastrium; in the right mammary line, it commenced at the sixth rib, and extended downwards about 4 centimètres (1·57 English inch); but the percussion tone was nowhere completely dull. The splenic dulness commenced at the eighth rib, but its lower margin could not be defined owing to the ascites and the œdematous condition of the integuments. The abdomen was greatly distended by a large quantity of liquid and gas, but was nowhere tender. The tongue was coated yellow and dry. After the administration of Tincture of Colocynth, the bowels were freely moved; the stools were of a brown color. Urine scanty, scarcely 300 cubic centimètres (10½ fluid ounces) in the day, dark reddish-brown, dense, with a reddish sediment of urates, and a small quantity of bile-pigment; it was free from albumen. Pulse 80.

On the 19th, pulse 84; respirations 22. The ascites and dyspnœa were rapidly increasing; less jaundice; urine more scanty, only about 200 cubic centimètres (about 7 fluid ounces), in 24 hours, turbid, neutral, and rapidly decomposing. The sediment contained a large quantity of globular lithates, also triple phosphates, and a few small octohedres of oxalate of lime. The patient complained of pricking pains in the region of the liver and was very morose. Was ordered Infusion of Rhubarb.

On the 22d, pulse 84; respirations 24. The hepatic dulness, close to the sternum amounted to 2 centimètres in the mammary line to 4, and in the axillary to 7 (·787, 1·57, and 2·75 English inches). Three stools daily, always tinged with bile. Appetite completely gone; cough less troublesome. Colocynth and Rhubarb were taken regularly.

On the 30th, pulse 100; respirations 26; headache and bilious vomiting. For eight days the jaundice had been diminishing, but the ascites had increased. The dyspnœa was more urgent, and the secretion of urine had almost entirely ceased.

On the 5th of May, paracentesis was performed, and about five pounds of a bright-yellow, perfectly clear fluid were drawn off from the abdomen; on the addition of strong nitric acid, this fluid threw down a bluish-green coagulum of albumen. After the tapping, small nodules could be felt in the epigastrium, and beneath the margins of the right ribs. About an inch and a-half farther down, there was an omental hernia about the size of an eight-groschen piece (13·458 English lines) in the abdominal parietes. Immediately after the tapping, the respirations

[1] Ohlau is a market-town in Silesia.—TRANSL.

became less embarrassed. In the afternoon, the patient only complained of slight giddiness. Was ordered to take Tincture of Rhubarb and Ether.

On the 6th, pulse 90; respirations 26; had slept well. A large quantity of serum was flowing from the punctured wound. The region of the liver was tender when touched. Stools very scanty; no urine passed.

On the 8th, pulse 84. The serum still continued to flow away. The tenderness over the abdomen was inconsiderable; in the hepatic region and at the site of the opening, it was less than before. Complained of nausea, but had no vomiting.

On the 11th, the serum still continued to flow away; no symptoms of peritonitis. Radial pulse very feeble, 84. Urine extremely scanty; only a few ounces in the day. Appetite improved.

On the 16th, pulse 100; increased tenderness of the abdomen. Since the day before, the wound had completely closed, and the abdomen had rapidly increased in size.

On the 17th, pulse 96; bilious vomiting, increasing collapse.

On the 19th, pulse 100; abdomen greatly distended, but not very tender; frequent vomiting and coldness of the extremities. The vomiting returned during the night; and, on the following morning, the patient died rather suddenly.

Autopsy.

Body extremely emaciated; no jaundice; no œdema of feet.

Dura mater somewhat thickened; a little thin blood in the longitudinal sinus; vessels of the pia mater slightly injected; patches of opacity in the arachnoid; brain pale and bloodless, of normal consistence.

Mucous membrane of the pharynx, œsophagus and air-passages normal; thyroid gland enlarged and infiltrated with colloid matter (*Colloidmasse*). Old adhesions over some parts of lungs; the lower lobe of the right lung in particular firmly adherent to the diaphragm. Left lung emphysematous and anæmic; hypostasis of the posterior and inferior parts of both lungs. Two drachms of fluid in the pericardium. Heart covered with a thick layer of fat; a large quantity of firmly coagulated blood in the right ventricle; loosely-coagulated blood in the pulmonary artery and in the left ventricle. Valves and muscular tissue of the heart normal.

Four pounds of yellow, tolerably clear fluid in the abdominal cavity. Spleen 5½ inches long, 3¾ inches broad, 1 inch thick, flabby, reddish-brown, and infiltrated with bluish-black pigment. Omentum somewhat thickened. A hernial sac, the size of a walnut, into which portions of the omentum extended, was found in the abdominal wall, close to the insertion of the ligamentum teres. Mucous membrane of the stomach smooth, at some places livid and covered with a thick layer of gray mucus; superficial cicatrices in the duodenum. Pancreas normal. The mucous membrane of the cæcum and of the ilium was livid; dirty-brown patches were observed above the ileo-colic valve, extending upwards into the ileum; and higher up, were blue cicatrices corresponding in form and situation to Peyer's patches. Fæces pale.

Kidneys anæmic and of normal consistence; bladder healthy. The serous covering of the anterior surface of the uterus and broad ligaments was much injected, and covered with puriform exudation. The right ovary contained several cysts the size of a nut, and was adherent to the Fallopian tube. The uterus was small.

Liver small and shrivelled. A tight-lace furrow was observed on the right lobe. The parenchyma presented everywhere a granular character, and a dense, firm, leathery consistence. The granulations varied in size from a pin's head to a linseed, and were separated by correspondingly narrow rims of areolar tissue. The bile was scanty and pale, and contained a large quantity of mucus.

Observation No. XXVII.

Intermittent Fever of seven months' duration.—Ascites.—hydræmia.—Anasarca.—Death from Œdema of the Lungs.

Autopsy.—A moderately enlarged Pigment-spleen.—Cirrhosis of the Liver—Mucous Membrane of the Stomach and Intestines, and likewise the Kidneys, normal.

Meta Horn, aged 51, was admitted into the Academical Hospital at Kiel, on the 28th of July, 1851. Her skin was of a pale, dirty-yellow color, and her muscles flabby and shrivelled, and her face and lower extremities swollen and œdematous. The thoracic organs presented nothing abnormal, with the exception of anæmic murmurs over the heart and great vessels of the neck; pulse 75 and soft. The abdomen was distended and yielded a dull percussion sound as far as the umbilicus, with distinct fluctuation; spleen somewhat enlarged; liver of normal size, and could be felt on palpation; appetite unimpaired; bowels costive; urine pale and free from albumen.

The patient stated, that seven months before, she had begun to suffer from intermittent fever, which for the first three months was of a tertian, and afterwards of a quotidian type. At the beginning of May, the fever had ceased, and at the same time the abdomen had begun to swell.

Was ordered to take the Ethereal Tincture of the Muriate of Iron, beef-tea and wine.

The patient, who had previously been greatly neglected, and who, on admission, was in a state of extreme exhaustion, did not rally. Two days after admission, symptoms of acute pulmonary œdema set in, under which she died.

Autopsy.

There was nothing abnormal in the cranial cavity, with the exception of great anæmia of the brain and its membranes.

The mucous membrane of the bronchi was pale and covered with frothy mucus; both lungs were œdematous, and the pleural cavities contained several ounces of clear serum. The valves of the heart, normal; its muscular tissue, flabby.

About nine pounds of pale-yellow serum were found in the peritoneal cavity. The stomach was small and contracted; its mucous membrane was pale, except at a few places where it was injected; the mucous membrane of the small intestine was very vascular; the cæcum and the colon contained firm, brown fæcal matter.

The uropoietic and genital organs presented no morbid change of importance.

The spleen was moderately enlarged, tolerably firm, of a reddish-brown color, with blackish pigment deposited at some places.

The liver was adherent at several places to the diaphragm; and the gall-bladder adhered to the transverse colon. The liver was but slightly reduced in size; its outer surface was granular, and its consistence very firm. On section, there were observed brownish-yellow granulations surrounded by pale rims of areolar tissue, which were most developed in the left lobe. Bile thin and pale.

Observation No. XXVIII.

Old Pleuritic Exudation.—Persistent Intermittent Fever.—Tubercle of both Lungs.—Ascites.—Bronzed Skin.—Small Liver.—Dyspeptic Symptoms.

Autopsy.—Firm Adhesions of Pleuræ.—Tubercle of the Lungs.—Cirrhosis of the Liver.—Supra-renal Capsules normal.

August Schunke, aged 70, was admitted into All Saints' Hospital on March 22d, 1858; three years before, he had been treated in the same Hospital for an exudation into the left pleura, from which he had recovered with flattening of the thorax. He was afterwards readmitted on account of an attack of intermittent fever, which was only cured after a protracted use of Quinine and Muriate of Iron. The patient was known to have been addicted to drink brandy.

On admission, he complained that some weeks before he had felt hoarse and began to cough and expectorate; and that soon after his recovery from the intermittent fever, his abdomen, and afterwards his feet, had become swollen. He was extremely emaciated; the skin was of a bronzed hue, and the conjunctivæ white. There were the physical signs of infiltration in the apices of both lungs; and, on the right side, of a cavity; sputa copious, yellow, and, in part, nummular. Pulse 80, and small; sounds of the heart normal. Hepatic dulness diminished; that of the spleen moderate in extent. There was fluid effusion in the abdominal cavity extending as high as the umbilicus; the abdominal veins were not enlarged. Tongue covered with a gray coat; appetite bad; bowels regular; stools brown. Urine scanty, free from albumen and bile-pigment.

At first, the patient was treated with Infusion of Rhubarb and Liquor Ammoniaci Anisatus,[1] and subsequently, with Extract of Cinchona and other tonics, together with an easily-digested animal diet. Notwithstanding, he became rapidly more exhausted, and on May 26th died.

Autopsy.

Recent injection of the larynx and trachea; the mucous membrane over the arytenoid cartilages slightly œdematous. Left lung firmly adherent by a thick membrane; upper lobes of both lungs infiltrated with tubercle, a cavity the size of a hen's egg in the apex of the right lung. Heart small; its valves normal; extensive atheromatous deposit in the coats of the aorta.

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[1] See note, p. 92.

About five pounds of clear serum in the abdominal cavity. Mucous membrane of the stomach tumid, and, in the neighborhood of the pylorus, of a bluish-gray color; several tubercular ulcers in the ileum. Spleen of moderate size, tolerably firm, and dark-brown. Liver very small, uniformly granular, firm, tenacious, and of a yellowish-brown color. Two concretions of a mulberry-form in the gall-bladder.

Supra-renal capsules in every respect normal. The cortical substance of the kidneys presented several cicatrix-like depressions; but in other respects these organs were healthy.

The five following cases are examples of cirrhosis of the liver in persons affected with syphilis. In three of them, the organ presented distinct traces of constitutional syphilis, which gave a peculiar character to the cirrhosis. In the two others, No. XXIX. and No. XXXIII., the cirrhosis had been preceded by syphilis, but the appearances presented by the liver were not such as ordinarily result from constitutional syphilis; and in both cases it was ascertained that the patients had been addicted to the use of spirits. Hence, in these two cases, the syphilis must be looked upon as something accidental.

Observation No. XXIX.

Previous Syphilis.—Abuse of Spirits.—Double Pneumonia.—Death from Œdema of the Lungs.

Autopsy.—Inflammatory Exudation in both Lungs.—Cirrhosis of the Liver.—Moderate Tumefaction of the Spleen.—No Ascites, and no Gastro-enteric Catarrh.

Johanne Krause, a servant-maid, aged 28, was admitted on the 20th February, and died on the 23d of the same month. She had been taken ill on the evening of the 18th with an attack of rigors, followed by pricking pains in the chest, cough, and expectoration of reddish, tenacious sputa. From expressions dropped during her noisy delirium, it was suspected that she had previously been addicted to spirit-drinking, and, in addition to this, she bore the marks of having formerly suffered from syphilis.

There was dulness with a consonating respiratory murmur over the upper portion of the right lung, both in front and behind; over the upper portion of the left lung there was a somewhat muffled tympanitic sound on percussion, and indistinct respiratory murmur; over the lower portion of both lungs, the percussion sound was clear and the respiration puerile. Violent fever. Pulse 120. Sputa dark reddish-brown, and very tenacious. Infusion of Digitalis was prescribed.

During the night, the patient became restless; at the same time, the upper lobe of the left lung became infiltrated with exudation, and both lungs were attacked by œdema, for which benzoic acid was taken without any benefit. The patient died at six in the morning of the 23d.

Autopsy on the 24th.

Skull-cap normal; dura mater congested; blood in the sinuses partly fluid and partly coagulated; the dura mater glued to the arachnoid by

a thin layer of dry, gray exudation; the arachnoid and pia mater greatly injected, and their vessels tortuous and winding. Only a few drops of serum at the base of the brain. Cerebral substance much injected, and of normal consistence.

Thyroid gland normal; bronchial glands melanotic. Larynx and trachea of a rosy-red hue; lining membrane of bronchi dark-red; the upper lobes of both lungs glued to the surface of the chest by recent fibrinous exudation; both upper lobes non-crepitant, firm, and presenting at some places red, and at others gray, hepatization; the dependent portions of the lower lobes congested and œdematous.

A little clear serum in the pericardium. Heart of normal size, and covered with a thick layer of fat; its muscular tissue and valves normal.

No serous effusion was found in the abdominal cavity. The mucous membrane of the stomach and intestinal canal was pale, and free from any structural lesion. Omentum fatty. Mesenteric glands normal. Spleen large, 7 inches in length by 3½ inches in breadth, of normal consistence, and rather anæmic.

The fundus uteri was inclined towards the left, and adhered to the left ovary, which contained a large corpus luteum filled with a chocolate-colored pulp, likewise several smaller corpora lutea of a yellow color, and a few cysts filled with serum. The left Fallopian tube was congested, and contained bloody mucus. Right ovary atrophied; right Fallopian tube much dilated, tortuous, and filled with serous fluid. Mucous membrane of the uterus hyperæmic, and covered with bloody mucus.

The liver was adherent at many places to the neighboring organs. The left lobe was larger than the right; both lobes, on the upper convex, as well as on the lower concave, surface, were very nodular, the nodules varying in size from a pea to a cherry. The serous envelope was thickened, and marked by cicatrix-like depressions. The consistence of the organ was tenacious and leathery. Bile dark.

Observation No. XXX.

Constitutional Syphilis.—Repeated courses of Mercury.—Albuminuria.—Splenic enlargement.—Right Pleurisy.—Dropsy.—Death from Acute Enteric Catarrh.

Autopsy.—Amyloid degeneration of the Kidneys, Spleen, and Liver.—Cirrhotic Shrivelling and Lobulation (Lappung) of the Liver.—Purulent effusion in the Right Pleura.—Cicatrices and old Ecchymoses of the Stomach.—Catarrhal Inflammation of the Small Intestine.

Rosine Conrad, a day-laborer, aged 36, was admitted on the 25th January, 1856, and died on the 9th February. The patient had previously suffered on several occasions from the symptoms of primary and secondary syphilis, for which she had often been a patient in the syphilitic department of the Hospital, where she had gone through several courses of mercury.

For two years, the patient had been known at the Polyclinique, where she had been treated several times for albuminuria and anasarca; and on each occasion she had gone away improved. On admission, she was free from œdema, and her appearance was tolerably healthy. For fourteen days,

she had complained of a slight cough without much expectoration; four days before admission, she had been seized with rigors, pain in the right side, and great dyspnœa. The right half of the thorax, as high as the third rib, was dull on percussion, and yielded no respiratory murmur; posteriorly, close to the spinal column, the dulness was less marked, and there was rough vesicular breathing with moist râles. The liver was pressed downwards, and the heart displaced towards the left. The exudation increased with tolerable rapidity; the dulness gradually extended at the lower and back part, but over the apex the respiratory murmur continued audible, and the sound on percussion was clear. The urine, which was passed in moderate quantity, was so loaded with albumen, that on boiling it became almost solid. Moderate fever; slight diarrhœa; no appetite. The use of Acetate of Ammonia, Dover's Powder, and warm baths, certainly succeeded in producing a constant secretion from the skin, and in effecting some diminution in the amount of albumen in the urine; but, notwithstanding, the anæmia rapidly increased, and the lower extremities became œdematous.

From the 2d of February, the symptoms were: nausea, repeated vomiting, light, very watery stools, and collapse, from which the patient could not be roused, notwithstanding the administration every hour of wine, and from 10 to 15 drops of the Liquor Ammoniæ Anisatus.[1] The stools became white and resembled whey. These symptoms were followed by præcordial uneasiness, cold extremities, imperceptible pulse, and, ultimately, for some hours, by delirium.

Death occurred at 11 o'clock on the morning of the 9th of February.

Autopsy on the 11th.

Skull-cap smooth, without any elevations, or loss of substance. Blood in the sinuses of the dura mater loosely coagulated; cerebral membranes injected; brain-substance firm and dry; gray matter congested.

A few condylomatous excrescences, the size of oat-seeds, upon the uvula; but no other local remains of syphilis.

Larynx normal; trachea and bronchi moderately congested. Left lung loosely adherent; its parenchyma normal, but at the lower and back part congested and somewhat œdematous. The right side of the thorax was filled up to the top in front with a purulent fluid; the liver was displaced downwards, and the heart was pushed to the left beyond the left mammary line, its apex lying beneath the fifth rib. The right lung was firmly connected posteriorly by old adhesions to the vertebral column, over an extent of two inches; its parenchyma at this place contained air at some parts, and at others were non-crepitant; the bronchi were filled with a quantity of muco-purulent secretion; the anterior portion of the lung was everywhere condensed and of a bluish-gray color. The pericardium contained a small quantity of serum; the valves and muscular tissue of the heart were normal.

On the mucous surface of the small curvature of the stomach was a radiated cicatrix, which produced a distinct constriction (*Abschnürung*), between the cul-de-sac and the pyloric portion; besides this, there were brownish-gray submucous extravasations of blood of an old date. The

[1] See note, p. 92.

serous covering of the small intestines was injected, and of an uniform rose-red hue; the mucous membrane was also very vascular, and the solitary glands prominent; the lower portion contained a reddish-gray fluid.

The larger vessels of the large intestine were injected.

Pancreas firm, mesenteric glands partly calcified. Spleen large, firm, brownish-red, very lardaceous, and containing glistening bodies like sago-grains.

Kidneys large; capsule separable; the parenchyma tinged yellow, partly firm and lardaceous, and partly friable (*frischbrüchig*).

The urinary bladder contained no urine: its mucous membrane normal.

Uterus and ovaries adherent to the neighboring organs: cysts in the Fallopian tubes; great omentum adherent to the fundus uteri; parenchyma of the uterus normal. A recent corpus luteum in the right ovary: (the menses had appeared on January 27th). Vagina smooth; small losses of substance with hæmorrhagic margins and base in its lining membrane. Labia majora very callous and marked by cicatrices and pigment-spots. Several white, radiated cicatrices, as if from the inoculation of matter from a chancre, were observed upon the right thigh.

The liver was everywhere firmly adherent to the diaphragm; the left lobe was completely atrophied and looked as if it were blended with the diaphragm; on the upper part of the convex surface were deep cicatrix-like depressions, enclosing portions of the hepatic tissue about the size of a walnut; the whole parenchyma nodulated, very firm, glistening and reddish-brown. The bile thick and of a mucous, almost gelatinous consistence, dark, depositing a quantity of coloring-matter, but containing no albumen.

Observation No. XXXI.

Constitutional Syphilis.—Systolic Bruit over Apex of Heart.—Dyspnœa —Cyanosis.—Large Spleen.—Bulging, nodulated Liver.—Albuminuria.—General Dropsy.

Autopsy.—Incompetence of the Mitral Valves.—Lardaceous Degeneration of the Liver, Spleen, and Kidneys.—Cicatrices and Cirrhotic Degeneration of the Liver.—Obliteration of a portion of the Portal Vessels.—Remarkable increase of the white Blood-corpuscles in the Portal and Hepatic Veins.

Johanne S——, aged 42, a female who had repeatedly been treated for syphilis, was admitted into All Saints' Hospital at Breslau, on April 20th, 1854.

She was of a pale, cyanotic appearance, had general dropsy, and complained of great dyspnœa, palpitations of the heart, and pains in the region of the liver. A loud systolic bruit was audible over the apex of the heart; the transverse cardiac dulness was considerably increased. There was a moderate amount of effusion in both pleural cavities, and a very considerable amount in the peritoneum. Urine scanty, pale-yellow, and very albuminous. The right lobe of the liver extended beyond the margin of the ribs, and could be felt on palpation; its margin appeared

rounded, its consistence was firm, and its surface nodular, and, at some places, even lobulated. There was no dulness in the epigastrium. Spleen considerably enlarged. For a long time the digestive powers had failed, and the patient had been going about quite neglected; for three weeks she had suffered from diarrhœa.

The dyspnœa and cyanosis rapidly increased and were accompanied by the expectoration of a serous fluid; the extremities were cool; but still the consciousness remained unimpaired. Death occurred early on the 22d, being preceded for some minutes by a continuous attack of general convulsions.

Autopsy, 18 *hours after death.*

On the frontal bone were observed the remains of old syphilitic disease. Brain and cerebral membranes normal.

Both pleural sacs contained several pounds of serous fl id; the pericardium contained about ten ounces, and the periton um a large quantity. The fluid in the peritoneum was pale-yellow, and deposited everywhere a number of gelatinous fibrinous coagula of a yellow color. Lungs œdematous, firm, and loaded with pigment. The margins of the mitral valves thickened and shrivelled; the right side of the heart enlarged and hypertrophied.

Mucous membrane of the stomach and intestine pale. The mesenteric and inguinal glands enlarged and infiltrated with gelatinous matter.

Both kidneys about one-third larger than in the normal state; cort'cal substance pale-yellow; epithelium fatty; the vascular loops of the Ma pighian capsules in a state of lardaceous degeneration. Spleen large, firm, and glistening like wax.

The liver weighed 2·2 kilogrammes (4 pounds 13½ ounces avoird.); its right lobe was remarkably enlarged and covered with cicatrix-like depressions, which enclosed nodules varying in size from a hazel-nut to a hen's egg, and, at some places, coarsely granular; the left lobe was shrivelled into a small nodulated appendage of tenacious, leathery consistence. (See Fig. 6.) The cut surface of the gland presented stripes of connective tissue of greater or less bread.h, which divided the yellow, waxy-looking parenchyma, at one place into large, and, at another, into small, islets, and which imparted to the whole a very firm consistence.

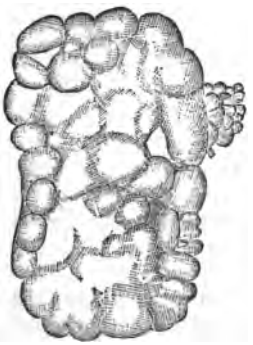

Fig. 6.—Liver affected with waxy and cirrhotic degeneration. The right lobe much enlarged and no ulated; the left, shrivelled into a small, nodulated appendage.

The sheath of the portal vein was considerably thickened; the blood found in this vessel contained nearly as many white as red blood corpuscles. The blood of the hepatic veins also abounded in white corpuscles. The larger branches of the portal vein were at some places narrowed and angular; the smaller twigs contained a dirty-brown blood-clot of old date, and some of them were obliterated. The lymphatic glands of the *porta hepatis* were enlarged and lardaceous.

Syphilitic cicatrices were observed at the entrance to the vagina.

Observation No. XXXII.

Constitutional Syphilis.—Hæmoptysis.—Dulness and consonant râles over the apex of the left Lung.—Ascites.—Albuminuria.—Tenderness and slight dulness in the region of the Liver.—Thin, pale stools.

Autopsy.—Small, Cirrhotic, Indurated Liver.—Moderately large Lardaceous Spleen.—Syphilitic Disease of the Cranial Bones.—Tubercle at the apices of both Lungs.—Granular Kidneys.

Johanne Kuehnemann, the widow of a joiner, aged 52, was admitted on June 4th, 1855, and died on February 3d, 1856.

In former years, she had suffered repeatedly from syphilis; a sunken nose, together with frequent attacks of periosteal affections, rapidly getting well under the use of Iodide of Potassium, indicated the existence of constitutional syphilis.

For several weeks the patient had been troubled with a cough, and three months before admission she had an attack of copious hæmoptysis. Dulness and consonant râles over the apex of the left lung; quick vesicular breathing, with prolonged expiration over right apex; the lower portions of both lungs normal.

Hepatic dulness diminished; frequent pain, increased by pressure, in the right hypochondrium. Owing to the existence of ascites, the spleen could not be felt. A moderate quantity of albumen in the urine; œdema of the extremities; extreme anæmia; slight febrile disturbance; tendency to diarrhœa; appetite moderate.

An animal diet and Extract of Peruvian Bark dissolved in Fennel-water were prescribed.

At a later date, the albuminous contents of the urine increased, and a few pale fibrinous casts (*blasse Cylinder*) could be detected in it; the œdema increased; but the chest-symptoms remained unchanged; the stools were pale and of little consistence. Gradually the strength failed; and ultimately the serous fluid oozed through the skin, while at the same time the patient became rapidly collapsed, and died on the 3d February, 1856.

Autopsy, on February 5th, 43 hours after death.

Skull-cap thick and condensed; flat ivory-like prominences on its inner surface close to the median line. Dura mater opaque, and thickened along the longitudinal sinus; firmly-coagulated blood in the longitudinal sinus; arachnoid grayish-white and thickened; pia mater moderately congested; consistence and vascularity of cerebral substance normal.

The mucous membrane of the larynx pale; that of the trachea and bronchi somewhat injected. Thyroid gland small and congested; calcareous deposit in some of the bronchial glands. A small quantity of serous fluid in both pleural cavities. The upper lobe of the left lung was firmly adherent, and infiltrated with gray tubercular matter; and, in addition to this, there was a recent gelatinous-looking exudation, through which the miliary tubercles were interspersed: at the apex, there was a cavity the size of a hen's egg with smooth, hyperæmic walls. The lower lobe was congested and contained recent miliary tubercles. The upper lobe of the

right lung was firmly adherent and contained a mass of yellow tubercle the size of a pigeon's egg.

Two ounces of clear fluid in the pericardium; patches of opacity (*Sehnenflecken*) over the right ventricle; margins of mitral valves somewhat thickened; heart normal in all other respects.

The mucous membrane of the stomach pale; in the small intestine, near the ileo-colic valve, was a small, excavated, recent ulcer, and yellow tubercle beneath the mucous membrane; pale, pulpy, fæcal matter in the colon and cæcum, the mucous membrane of which was of a slaty-gray hue.

The spleen was slightly enlarged, very lardaceous, dry, and reddish-brown; Malpighian bodies not visible.

Liver remarkably small; its right lobe completely hidden by the tortuous curvature of the ascending colon. Surface of the liver uneven and covered with nodules from the size of a pea to that of a linseed; its margins shrivelled; parenchyma nodulated and firm. In the convex portion of the right lobe was a cyst, the size of a walnut, containing dead echinococci. The liver was connected to the diaphragm and the adjoining organs by numerous bands, which were infiltrated with serum. Bile dark and inspissated.

Surface of kidneys slightly granular and capsule firmly adherent; whitish infiltrations of firm consistence in some portions of the cortical substance. Bladder normal.

The ovaries and Fallopian tubes were adherent to the posterior wall of the uterus, which was atrophied; cicatrices in the vagina. A very red excrescence, like a cock's-comb, was observed above the orifice of the urethra.

Observation No. XXXIII.

Constitutional Syphilis.—Epilepsy.—Abuse of Spirits.—Death in an Epileptic Fit.

Autopsy.—Cirrhosis of the Liver.—Enlarged Spleen.—Chronic Catarrh of the Stomach.

Julius Kessel, laborer, aged 38, was admitted into the syphilitic department on the 7th of April, 1858, and on the 8th, died in an epileptic attack. He was a drunkard, and, several years before, he had had an attack of primary syphilis; on admission, he was found to be suffering from ulcers in the pharynx, pains in the bones of the head, and tibia, and syphilitic rupia. He had been afflicted with epilepsy for a still longer period. No other information was obtained respecting his previous history.

Post-mortem appearances.

At the base of the cranium, the left posterior clinoid process was found to be very prominent, and furnished with a sharp angular point, whereas the right process was flat and its surface smooth. The portion of brain corresponding to the left process was somewhat softened. There was nothing else abnormal in the cranial cavity.

The thorax was narrowed from lateral curvature; the lungs were

congested and œdematous. The right side of the heart was enlarged, its muscular tissue, particularly on the left side, was pale and friable.

There was no fluid in the abdomen. The spleen was enlarged, soft, and loaded with blood. The mucous membrane of the stomach was in part recently injected and tumid, and in part, especially near the pylorus, grayish-brown and thickened. The coats of the small intestine were at some places intensely injected, and at other places paler. The mucous membrane in the reddened portions was covered with bloody fluid. The mesenteric veins were not enlarged to any great extent. The fæces were pale, and of normal consistence.

The kidneys were of normal consistence; the right, large and congested, and the left, small. Urine straw-colored, and free from albumen.

The liver was much shortened in its long, but scarcely in its transverse, diameter; its surface was uniformly granular, and adherent at many places to the surrounding organs. On section, its surface was found to be finely-granular and reddish-gray; the veins were enlarged, and the hepatic artery remarkably so.

The following case is of great interest, owing to the extensive colloid or lardaceous degeneration of the organs, which had produced an enormous enlargement of the liver and spleen. An intense degree of jaundice was induced in this case, by the colloid infiltration of the lymphatic glands in the *porta hepatis*. The trifling derangement of the patient's nutrition, notwithstanding such extensive degeneration of the liver, spleen and lymphatic glands, was remarkable. No particular cause could be discovered to account for the origin of the acute peritonitis.

Observation No. XXXIV.

Jaundice of 18 *months' duration.—Enlarged Liver with uneven surface. —Death under symptoms of Acute Peritonitis.*

Autopsy.—Granular Lardaceous Liver.—Lardaceous Spleen.—Infiltration of the Glands in the Fossa Hepatis, and in the Inguinal region. —Purulent Peritoneal Exudation.

Franz Gaida, householder, aged 50, was admitted on the 9th, and died on the 19th of November, 1852. The patient, who was a large, corpulent man, had suffered for a year and a-half from jaundice, pains in the region of the liver, and constipation. He had already, in the previous April, been for some time in the Hospital, and, after the protracted use of Rhubarb and Carbonate of Soda, the jaundice had in a great measure disappeared, but not entirely. At that time the left lobe of the liver extended to within one inch of the umbilicus, and the right lobe projected an inch beyond the margin of the ribs.

Present State:—Intense jaundice; urine deeply tinged with the coloring-matter of bile; bowels confined; stools white; appetite good; acid eructations; general strength still tolerably good; nothing abnormal in the respiratory organs or in the heart. The left lobe of the liver could be felt in the epigastrium, hard and covered with numerous nodules the size of a pea; it extended almost to the umbilicus. The right lobe projected to a

less extent. The dulness in the splenic region measured 7 inches in length by 5 inches in breadth.

The patient was ordered to take Infusion of Rhubarb with Carbonate of Potash; and under this treatment there was comparative improvement up to the 18th.

At midday of the 18th he was seized with rigors, vomiting of food, and burning pains at the epigastrium, followed by heat of skin and increased frequency of pulse. The abdomen was distended and tender upon pressure. Great uneasiness. Cataplasms and Morphia were prescribed. Towards evening he again vomited several times a bilious mucous substance, and passed four thin stools. A rapidly increasing collection of fluid could be detected in the lower part of the abdomen; extremities cold; pulse imperceptible. Death on the 19th, at 6 A.M.

Autopsy on November 20th.

The skull-cap was of a dark-yellow color, and the diploë intensely injected. Dura mater tinged pale-yellow; about an ounce and a-half of serum at the base of the cranium; pia mater moderately congested. The cerebral substance was somewhat softer than natural and presented a glistening appearance on section.

Mucous membrane of the larynx and trachea injected, somewhat relaxed, and covered with gray mucus. Thyroid gland healthy. Old adhesions of the right pleura; no fluid effusion. Great hypostatic congestion at back part of both lungs, which, however, were everywhere crepitant. The heart was enlarged transversely; the left ventricle contained tarry blood; the valves were healthy and deeply stained. Slight atheromatous degeneration of the coats of the vessel above the aortic valves. A large amount of turbid, somewhat viscid, deep yellow fluid escaped from the peritoneum, and a quantity of puriform fibrinous matter was collected in the pelvic cavity. The liver extended far down into the epigastrium; its surface was covered with numerous nodules, which in some places were collected in groups; deep cicatrix-like depressions were observed in other situations. The under surface of the liver, and particularly that part in the neighborhood of the gall-bladder, the stomach and the transverse colon, the duodenum, and the lesser omentum were all firmly united into one mass. The entire liver measured 12 inches from right to left; and the left lobe 7 inches. The margin of the right lobe was sharp, and its measurement from before backwards was 9¼ inches. A considerable mass of greatly enlarged, reddish-brown glands, presenting a grayish-white surface on section, was found in the *porta hepatis;* this mass compressed the bile-ducts, which in other respects were normal, except that they were dilated on the side of the enlarged glands next to the liver. The gall-bladder was not enlarged; it was filled with viscid, mucous, yellow bile, and likewise contained a few small, bluish gall-stones; the mucous membrane was somewhat relaxed. The nodules on the surface of the liver were as large as a bean; the parenchyma presented a greenish-yellow, lardaceous, glistening aspect, and grated under the knife. On the cut surface, broad stripes of connective tissue were seen enclosing insulated masses of the infiltrated parenchyma, varying in size from that of a pea to a hazel-nut.

The spleen was 7½ inches long and 5 inches broad; its capsule was a line and a-half thick. Its parenchyma was firm, lardaceous, and reddish-brown.

Kidneys rather large, flabby, soft, and congested. Pancreas thickened and dense, but free from infiltration. Retro-peritoneal and mesenteric glands unchanged.

Pharynx and œsophagus normal. The stomach contained undigested food; the mucous membrane near the pylorus was thickened and of a livid-gray hue; in the fundus it presented *post-mortem* softening. There was nothing abnormal in the intestinal canal. The mesentery was loaded with fat.

The large vessels of the abdomen were normal; the iliac veins contained loosely-coagulated blood of a dirty hue.

The lymphatic glands in the fold of the thigh were enlarged and glistening from lardaceous deposit. The urinary bladder was healthy. There were no traces of former syphilitic disease upon the penis.

The following Observation resembles No. XXIV. Here also the disease of the liver was induced by chronic peritonitis, which involved the capsule of the liver, and from this extended into the parenchyma, giving rise to induration, and a lobulated condition of the gland.

Observation No. XXXV.

Abdomen enlarged and painful.—Deranged Digestion.—Ascites.—Enlargement of the Spleen.—Surface of the Liver felt covered with nodules. — Paracentesis. — Profuse watery Diarrhœa. — Exhaustion.— Death.

Autopsy.— Lobulated Cirrhotic Liver. — Enlarged Spleen. — Mucous Membrane of the Stomach and Intestines livid and much relaxed.

Rosalie Kassner, a tailor's wife, aged 43, was admitted on 23d of February, 1857.

For several years she had suffered from pains and distention of the abdomen, accompanied by loss of appetite, constipation, and occasional vomiting, and consequently she had become weak and emaciated. The menses had ceased a year before. On admission, ascites was discovered extending as high up as the umbilicus; and there had been slight œdema of the feet for a fortnight. The liver appeared to be reduced in size, so far as the tympanitic condition of the intestines, which were displaced upwards, would allow an opinion to be formed upon the matter; its dulness at the sternum and in a line with the right nipple did not exceed 3 centimètres (1·18 English inch). The spleen, on the other hand, was enlarged; it projected about 6 centimètres (2·36 English inches) beyond the false ribs, and extended over four intercostal spaces. Heart and lungs normal. Urine scanty and red, but free from albumen. Skin pale, and devoid of any jaundiced tinge.

Tincture of Colocynth was prescribed.

On the 24th, two pale stools; the whole abdomen distended and tender upon pressure; no appetite; tongue clean; pulse 84. The patient was ordered to take Infusion of Rhubarb with Ethereal Tincture of Valerian.

After this the œdema of the feet disappeared, but the ascites increased; the veins on the abdomen became greatly enlarged; two pale stools were passed daily; and the appetite was very slight.

On the 2d of March, the great tension of the abdominal wall and the dyspnœa rendered it necessary to have recourse to paracentesis, by means of which a large quantity of clear pale-yellow fluid was drawn off. Immediately after this the size and form of the liver could be determined with greater accuracy. The organ had sunk down, and could now be felt on palpation through the thin, flabby abdominal walls. Nodules, of greater or less size, and tender upon pressure, could be distinguished over its surface, particularly on the left lobe. Now that the gland had regained its normal position, percussion yielded entirely different results from what it had before the performance of paracentesis; the dulness close to the sternum amounting to 14 centimètres, in a line with the nipple to 16, and in the axilla to 15 (5·5, 6·29, and 5·9 English inches). The enlargement of the spleen likewise could be made out more easily; its rounded edge could be felt 7 centimètres (2·75 English inches) below the margin of the ribs.

On the 3d of March, the patient felt relieved, and passed copious grayish-yellow stools; appetite slight. Compound Tincture of Cinchona was prescribed.

On March 4th, profuse diarrhœa; stools like rice water; great prostration and apathy; pulse 108; respirations 12. Was ordered to take Extract of Logwood in Cinnamon Water.

On March 7th, diarrhœa abated; pulse 72; respirations 10; collapse; skin cool.

The appetite failed completely, and the prostration increased more and more; from time to time, the diarrhœa returned; the abdomen continued painless and flabby; the pulse, which varied between 76 and 84, became smaller and softer, until, on March 11th, hiccough and tracheal râles set in, which terminated in death by exhaustion.

Autopsy, 25 hours after death.

Neither the brain nor its membranes presented anything abnormal.

The lining membrane of the bronchi was slightly injected; the lungs were œdematous; the pleural cavities contained a few ounces of serum. The heart was small; its valves and muscular tissue normal.

A few pounds of clear yellow fluid were found in the abdominal cavity. The peritoneum was pale; the mesentery at some places thickened; some of the mesenteric veins enlarged; mesenteric glands normal. The mucous membrane of the stomach was tumid and of a livid hue; the lining membrane of both the small and large intestine was dark, at some places blush-black and much relaxed, but without any loss of substance. The kidneys, urinary passages, and genital organs presented nothing worth recording. The spleen measured 6½ inches in length, by 4½ inches in breadth, and 1½ inch in thickness; it weighed 0·55 kilogramme (17¼ ounces avoirdupois); its parenchyma was pale-red, hard, and firm.

The liver was adherent at many places to the surrounding parts, and its form was remarkably altered. (Fig. 7.) A deep furrow passed transversely across the organ, dividing it into two halves. The left lobe was covered with nodules, varying in size from a pea to a walnut, and posteriorly it was marked by numerous deep fissures. The right lobe presented posteriorly a globular portion of healthy parenchyma, the surface of which was only marked by a few grayish-white cicatrix-like depressions; anteriorly, however, in front of the tight-lace fissure, were several easily-mov-

able, knotty protuberances, varying in size from a hen's egg to that of the fist. Numerous protuberances, separated by deep fissures, were likewise observed upon the under surface. The diameter of the right lobe, from before backwards, amounted to 7½ inches, and that of the left to 3; the transverse diameter of both lobes measured 4 inches. On further examination it was ascertained that the substance of the gland was traversed by broad bands of connective tissue, dividing it into numerous lobes and lobules.[1] In the parts occupied by the bands of connective tissue the secreting substance of the gland had completely disappeared, while the lobules which they surrounded consisted partly of healthy glandular tissue, loaded with pigment, and partly of cirrhotic or uniformly indurated tissue. Groups of three, five, or eight lobules of the liver were seen separated from one another by broad rims of connective tissue, in which the remains of the destroyed glandular substance and an intricate network

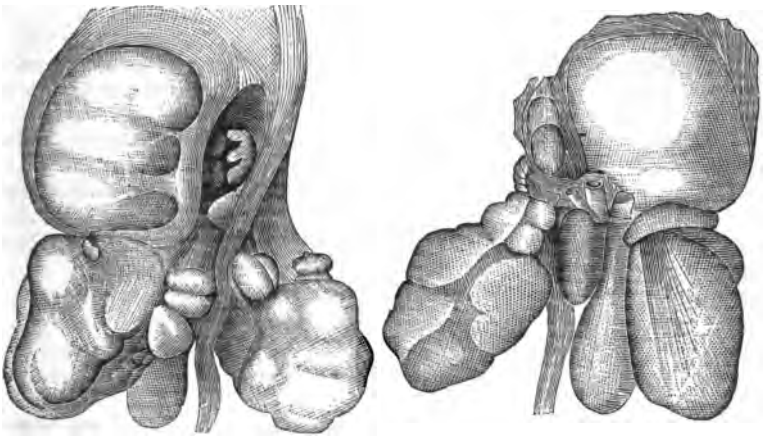

FIG. 7.—A lobulated, cirrhotic liver, with its form remarkably altered. The figure on the left hand represents the upper surface of the organ; that on the right hand shows the under surface.

of vessels could be detected. This network consisted, for the most part, of lengthened, very tortuous vessels, most of which could be injected from the hepatic artery, but some of them from the portal vein. The branches of the hepatic artery contained black pigment at many places. This vascular apparatus, as well as the connective tissue in which it was imbedded, was mostly of recent formation, and differed essentially from that of the normal liver in the distribution of its twigs and capillaries. The normal arrangement could only be discovered where the substance of the gland remained intact; in such places vascular meshes could be seen surrounding the hepatic cells, which could be injected partly from the portal vein, partly from the hepatic vein, and at some places from the hepatic artery.

Glisson's capsule was considerably thickened; the walls and calibre of the portal vein were normal. The hepatic artery was enlarged.

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[1] Strips of connective tissue passed from the capsule into the interior of the liver, separating individual lobules, or groups of these lobules, from one another.

The ascitic fluid consisted of:—

Water	97·84
Solid constituents	2·16
	100·
Albumen	1·18
Salts and extractive matter	0·98
	2·16

The following is another example of cirrhosis, which proved fatal under symptoms of dysentery.

Observation No. XXXVI.

Abuse of spirits.—Apoplectic attack.—During six years temporary enlargements of the Liver, accompanied by pain.—Jaundice.—Dyspnœa.—Bloody stools with tenesmus.—Slight somnolence.—Much albumen and kreatine and traces of the biliary acids in the urine.—Autopsy.—Cirrhotic induration of the Liver.—Hepatic cells partly destroyed.—Dysenteric disease in the small and large Intestines.—Pneumonia.—Cysticerci in the Brain and in the Thoracic Muscles.

Reuter, a tradesman, aged 46, was admitted into the Charité Hospital on December 31st, 1859.

Having always formerly enjoyed good health, he was seized in autumn, 1848, with an attack of apoplexy, and was paralyzed on the left side; from this date the left arm had always been weak. In 1853 he suffered from a painful swelling in the hepatic region, accompanied by dyspnœa. After two days this pain was removed by cupping, and, although it often returned, it always soon ceased, the dyspnœa, however, remaining permanent. There was no jaundice.

At the beginning of December, 1859, severe pains came on in both feet, which, after several days, were followed by œdema. Eight days after this jaundice made its appearance. The patient confessed to having been in the habit of drinking a considerable quantity of brandy.

On admission, the patient was a man of rather large build, but very much emaciated; his most comfortable positions were upon his back and right side; the skin and conjunctivæ presented a deep yellow tinge; the cheeks were livid-red; the tongue was red at the margins, with a grayish-white coat along the centre, and dry; appetite slight; great thirst; the sensorial faculties unimpaired. The lower extremities were tender upon moderate pressure or upon the slightest movement, and were somewhat œdematous. The abdomen was greatly distended, and contained a small quantity of fluid. In the right hypochondrium the lower margin of the liver could be distinctly felt, sharp and firm, through the thin abdominal walls; the surface of the left lobe was somewhat uneven and nodulated. The perpendicular hepatic dulness amounted to 18 centimètres in the mammary line, to 13 centimètres in the median line, to 10 centimètres from the base of the ensiform cartilage downwards, and to 8 centimètres at the left of the median line (7·086, 5·118, 3·937, and 3·149 English inches). The spleen was enlarged.

The cardiac impulse could be felt in the fifth intercostal space, a little to the inside of the mammary line. Over the apex of the heart there was a sinking inwards (*Einziehung*) accompanying the systole; here, moreover, a systolic blowing murmur, commencing with a musical note, was audible; the second sound was unusually abrupt. The cardiac sounds were of a similar character over the lower end of the sternum. Over the great vessels the first sound was rough, and the second, particularly over the sternal extremity of the sternum, was loud and flapping.

Infusion of Rhubarb and Senna Leaves was prescribed.

On January 1st, 1860, condition unchanged; a copious brown motion of the bowels; towards evening great restlessness, but, notwithstanding this, had quiet sleep during the night.

On January 2d, temperature 37·8° cent. (100·2° Fahr.); pulse 104; respirations 32. The pains in the feet had ceased; tongue moist, coated brown in the middle; no appetite; urine scanty, and dark reddish-brown. Towards evening the temperature rose to 38·4° cent. (101·12° Fahr.); pulse 92; respirations 18. Passed a restless night.

On the morning of the 3rd, temperature 37·8° cent. (100·2° Fahr.); pulse 96; respirations 22; the tongue had become clean; no appetite; three thin brownish stools; the œdema had disappeared from the feet; slight dry cough; a moderate amount of pain in the epigastrium. In the evening, temperature 38° cent. (100·4° Fahr.); pulse 84; respirations 24. Was ordered to take a quarter of a grain of Acetate of Morphia; slept well, and perspired a good deal.

On the morning of the 4th, temperature 37·5° cent. (99·5° Fahr.); pulse 120; respirations 24. The appetite had improved; tongue clean and moist; six thin, pale stools; urine scanty and dark. In the evening, temperature 38·2° cent. (100·76° Fahr.); pulse 116; respirations 24. Passed a quiet night after taking quarter of a grain of Morphia.

On the 5th, symptoms were the same as on the 4th.

On the 6th, complained of a feeling of numbness in the feet; the œdema had again increased; ten thin yellow stools, mixed with bright-red blood and a clot the size of an apple; tenesmus. Was ordered to take Phosphoric Acid and solution of Gum as a drink.

On the morning of the 7th, temperature 38·9° cent. (102·2° Fahr.); pulse 112; respirations 24. Consciousness was impaired; the jaundice had become more intense; the feet were still painful, and the œdema had increased. Slight cough, without any expectoration; percussion over the left side of the thorax yielded a tolerably clear sound at the apex, and a dull sound in the infra-spinous fossa as far down as the tenth rib. Similar results were obtained from percussion of the right side of the chest. Vesicular breathing was audible over the posterior and upper part of both sides of the chest; lower down, the breathing became more feeble, and was accompanied by fine moist râles. In front, the right side of the chest was clear on percussion; but, on the left side, there was slight dulness, together with fine, moist, crepitating râles. Tongue red and dry; abdomen slightly distended; about twenty pale-yellow, thin, very offensive stools, containing streaks of blood. Urine dark and loaded with pigment. Was ordered to have a starch enema.

During the night between the 7th and 8th of January, the patient was tolerably quiet, but his mind was confused. On the morning of the 8th, temperature 38·8° cent. (101·84° Fahr.); pulse 100; respirations 22. The pulse, which hitherto had been full and hard, became small and soft; copious fœtid evacuations; tongue red and moist; the swelling in the

hepatic region increased. In the evening, temperature 39·4° cent. (102·9° Fahr.); pulse 108; respirations 28. Was ordered to have a Tannin Clyster. During the ensuing night the patient suffered from restlessness, hiccough, and convulsive movement of the muscles of the trunk.

On the 9th, temperature 38·5° cent. (101·3° Fahr.); pulse 100; respirations 24. Stools scanty and free from blood. Towards evening involuntary motions of bowels. During the night great restlessness and slight delirium.

On the 10th, temperature 38·5° cent. (101·3° Fahr.); pulse 120; respirations 32 and panting; numerous thin stools, partly yellow and partly bloody. Urine brown and scanty.

Death occurred about noon of the 10th.

Autopsy.

Brain well developed; a large cysticercus in the left hemisphere, and also at the anterior extremity of the left lobe. The ventricles contained only a small quantity of fluid tinged with bile; their lining membrane was everywhere thickened; on the left side the walls of the posterior cornu had grown together; on the right side they were only slightly adherent. In the right ventricle, towards the anterior cornu, prominent warty and polypoid excrescences of a remarkable character were attached to the lining membrane. A few small cysts in the choroid plexus. Consistence of the brain everywhere firm. Parenchyma moist and glistening; numerous red puncta in the white substance; the gray matter firm, but pale. In the left hemisphere, close to the longitudinal fissure, was a cavity the size of a cherry-stone, with a dead cysticercus. Corpus striatum and optic thalamus, of a jaundiced tint, pale and firm. Cerebellum small and pale.

In the abdominal cavity there was a moderate amount of a bright-yellow fluid, mixed with recent gelatinous coagula. The intestines, and particularly the colon, were somewhat tympanitic. The intestinal contents were yellow in the jejunum, and lower down became greenish and flaky; in the ileum they were of a yellowish-brown color; the large bowel was empty except near its lower extremity, where there was a little reddish fluid. The folds of the mucous membrane of the rectum were tumid and reddened, and covered with a diphtheritic membrane. Higher up, the mucous surface of the bowel was partly superficially ulcerated, and partly covered with a recent diphtheritic exudation, the mucous membrane being destroyed, and presenting a pitted uneven appearance: this morbid condition increased in extent the higher we proceeded upwards. Some portions of the lining membrane of the colon and cæcum still remained sound; in the ileum the follicles were enlarged; the mucous membrane was thickened, and here and there very red; the folds in the jejunum were very tumid.

The stomach contained a large quantity of a mucous, dirty-yellowish fluid; its walls were much injected, and there were also a few minute ecchymoses, especially in the neighborhood of the pylorus. The orifice of the ductus choledochus was very prominent, and, upon pressure, a whitish plug was squeezed out of it. The gall-bladder was flabby, imperfectly filled, and œdematous, and poured out, upon pressure, a thin watery bile. The mucous membrane of the gall-bladder was very red and but slightly tinged with bile. The bile was reddish-yellow, and of a remarkably mucous character. The ductus choledochus was filled with pale bile, charac-

terized by feeble coloring properties; its lining membrane near to the orifice appeared normal, but the lips of the orifice itself were swollen and hyperæmic. The vena portæ contained a quantity of thin blood.

The liver was much enlarged and heavy. Transversely it measured 11½ inches, whereof 7⅛ inches belonged to the right lobe. The thickness of the left lobe was 2⅛ inches, of the right 3⅝ inches; the height (*i. e.*, from the lower margin to the most convex portion, the organ being still *in situ*) of the right lobe was 8½ inches, of the left 6¾ inches. Its surface was somewhat uneven; the capsule was thickened at some places and there were likewise flattened irregular prominences of the parenchyma. The color of the organ throughout was greenish-gray, intermingled with grayish-white spots, particularly in the right lobe; the left lobe was more of a red hue. The left lobe likewise presented a finely-granular character; its tissue was divided with difficulty, and exhibited, upon section, a granular aspect, the granulations having a diameter of from 1 to 1½ inch; along with these were grayish-white stripes, which at some places were almost like a cicatrix, homogeneous, and collected into whitish masses, in the interior of which no hepatic tissue could be detected. The hepatic cells were only at some places normal; at other parts they had become disintegrated, and their place was supplied by oil-globules and particles of coloring-matter.

The lobules of the pancreas were rather large and pale.

The kidneys were large and thick; 4¾ inches long, 2½ inches broad, and 1½ inch thick; their surface was smooth, with three small reddish depressions. On section they were found to be loaded with blood, with the exception of the papillæ, which were opaque white, and from which a yellowish fluid exuded on pressure. Cortical substance broad; glomeruli moderately filled; the tortuous uriniferous tubes very opaque. Suprarenal capsules large; their cortical substance atrophied; oily contents inconsiderable.

Scarcely any change in the lymphatic glands, except that those in the ileo-cæcal region were enlarged and red.

The costal cartilages were for the most part ossified; a dead cysticercus was found in the right pectoral muscles. Both lungs were much inflated and but slightly collapsed. Left lung large; bronchus full of mucous fluid; bronchial glands enlarged, and of a slaty color; the lung itself dense, containing a large quantity of pigment, presenting a jaundiced tint upon section, and moderately congested; extensive pneumonic exudation in the upper lobe and several masses of a similar exudation in the lower lobe. Right lung larger and firmer, very friable, and presenting an intense jaundiced tint upon section. The fluid scraped from the cut surface was very mucous and glairy; and the pulmonary tissue when squeezed was very œdematous, a reddish-gray tissue remaining after the separation of the fluid. The upper lobe was still perfectly crepitant; the greater portion of the lower lobe presented a condensed leathery appearance.

Thyroid gland rather small.

The pericardium contained a considerable quantity of fluid deeply tinged with bile. The heart was rather large, pale and fatty. It contained much fluid blood, together with a large deeply-jaundiced coagulum, which was very friable, and resembled the rind of bacon. The aorta was rather constricted; its valves were perfectly competent. There were granular vegetations upon the pulmonary valves, and all the valves presented an intensely jaundiced hue.

The glands in the anterior mediastinum, especially on the right side, were much enlarged and infiltrated with cheesy matter.

The urine was of a deep jaundiced tint, acid, and with a specific gravity of from 1020 to 1022. The quantity passed in 24 hours amounted, on January 3d, to 600 cubic centimètres, on the 4th to 700, on the 6th to 450, and on the 7th to 600. (21½, 24⅔, 15½, and 21⅓ fluid ounces apoth.). It contained urea, kreatine, kreatinine, and likewise uric acid in large quantity. In addition to these compounds, traces of the biliary acids were detected, and there was also found a quantity of a substance separating in the form of laminated, tabular crystals, the nature of which could not be determined, but which was perhaps sarcosine (?).

11. *Treatment.*

The indications for the treatment of cirrhosis of the liver vary according to the stages of the disease, and the consequences to which it gives rise. We have rarely an opportunity of observing the commencement of the affection or of treating its earlier stages, whilst it is still possible to arrest the degenerative process. In most cases, when the existence of the disease is recognized, this favorable period has passed away, and all that can be done is to treat the effects of the local disorder upon the functions and organs of digestion, and upon the general system,—an indication which can never be fulfilled save to a limited extent, inasmuch as it is impossible to remove the cause.

A painful swelling of the liver, accompanied by indigestion and other symptoms indicative of the approach of cirrhosis, is always to be treated with great care when it occurs in individuals who have been addicted to the use of spirits, or who have been suffering from diseases in the course of which cirrhosis is wont to be developed. Complete abstinence from spirits is indispensable in such cases; the diet must consist of mild, simple articles of nourishment, which, in strong persons, should be mainly of a vegetable nature, such as fruits, light pulses, and suitable farinaceous substances, but in debilitated individuals must consist of easily-digested animal food. Coffee, spices, and other articles, which irritate the liver, must be avoided.

When the swelling and tenderness are considerable, leeches are to be applied to the hepatic region and to the anus, mercurial ointment is to be rubbed in, and the right hypochondrium fermented with warm cataplasms. Internally we may prescribe mild saline laxatives in Decoction of Tamarinds or Grass-root (*Graswurzel*),[1] or in robust individuals, a few grains of Calomel; or the patient may drink bitter infusions, the saline Spring of Eger, the cold Karlsbad Spring,[2] &c. When the tenderness ceases, the bowels are to be kept open and the gastric digestion regulated, by means of Rhubarb, salines, and solvent extracts.

In cases where constitutional syphilis appears to be the primary cause, a regulated treatment by Iodide of Potassium or Iodide of Iron, or the use of the Adelaide Spring, or of the waters of Kreuznach and Baden; or of

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[1] The root of the *Triticum repens*. The decoction is said to contain sugar and free oxalic acid.—TRANSL.

[2] For the nature of the mineral waters of Karlsbad and Eger, see Vol. I., pp. 88 and 220, notes.—TRANSL.

Aix-la-Chapelle is to be preferred.[1] Under certain circumstances a treatment by means of small inunctions may be tried.

Intermittent fever, when possible, is to be removed by a protracted change of air; the hyperæmic condition of the liver which remains can then be treated according to the principles laid down in the first volume.

In most cases, where medical advice is applied for, the cirrhotic degeneration has already advanced so far that little can be done in the way of treatment. This should be restricted to checking the peri-hepatitis which supervenes from time to time, by means of rest, cataplasms, and, when necessary, by cupping. Care must then be taken not to combat the degeneration by means of powerful mineral waters, such as the warm springs of Karlsbad, or the laxative waters of Marienbad,[2] &c., or by mercury, iodine, or similar preparations; by such measures, the unfavorable termination is only hastened, owing to the increased exhaustion and the accelerated progress of the degeneration, as I have myself observed after the employment of the waters of Karlsbad. In such cases nothing remains to be done but to check, as far as possible, the consequences of the hepatic disease, which manifest themselves in the disorders of the digestive organs, the dropsy, the impaired nutrition, and the defective formation of blood.

The best remedies which can be employed for stimulating the functions of the stomach are the bitter medicines, such as the Tincture of Rhubarb, solvent extracts, Extract of Orange-peel, Wormwood (*Artemisia Absinthium*), &c., dissolved in an Aromatic Water, Infusion of Quassia, and the Root of Calamus Aromaticus, either by itself, or with the addition of Choleate of Soda. When nausea or vomiting is present, the bitter remedies ought to be combined with preparations containing Hydrocyanic Acid, or with small doses of Extract of Belladonna, Morphia, the Magistery of Bismuth, &c. In drunkards the aqueous extract, or the tincture, of Nux Vomica is particularly suitable. Hæmorrhage from the stomach may necessitate the employment of strong astringents, such as Tannin, Acetate of Lead, and Nitrate of Silver.

The bowels are to be regulated by Rhubarb, Aloes, Ox-bile, and similar remedies, and saline purgatives ought now to be avoided, as they are very apt to produce watery evacuations. The attacks of diarrhœa, which occur not unfrequently in the later stages of the complaint, are to be checked by Calumba, Cascarilla, Extract of Logwood, Nux Vomica, Tannin, &c. When there is great meteorism, it is well to add to the bitter or laxative remedies the Ethereal Oils: Anise Oil, Fennel Oil, and Cajeput Oil, or small doses of Ether; and, in addition to these, the abdomen may be rubbed with Eau-de-Cologne, the Mistura Oleoso-balsamica,[3] &c. The Choleate of Soda, dissolved in Infusion of Rhubarb, or in some aromatic water, operates exceedingly well in regulating the intestinal digestion,

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[1] These springs are for the most part thermal and saline. The principal mineral ingredient in all of them is the chloride of sodium. The thermal springs of Kreuznach, which contain the largest quantity of chloride of sodium, have a temperature of 88° Fahr. The temperature of the springs at Baden-Baden varies from 113° to 145° Fahr., that of the springs at Aix-la-Chapelle from 113° to 131° Fahr.—TRANSL.

[2] See Vol. I., p. 88, note.—TRANSL.

[3] The *Mistura oleoso-balsamica* is the *Balsamum vitæ Hoffmanni*. It is a solution, in rectified spirits, of numerous aromatics, such as oil of lavender, oil of cloves, oil of cinnamon, oil of thyme, oil of citron, oil of mace, oil of orange flowers, and balsam of Peru (1 part of the first seven ingredients, and 3 of balsam of Peru, to 240 parts of rectified spirit).—TRANSL.

and removing the meteorism. In intestinal hæmorrhages which threaten to prove exhausting, Tannin, both internally and in the form of enema, is to be recommended.

The ascites is always difficult to treat; so long as it does not exceed a moderate amount, we must forbear from all powerful remedies, because the injury which the digestive organs may sustain from diuretics or drastic purgatives exceeds any advantage which might be derived from a temporary removal of the ascites. Diuretics are rarely of much effect against the ascites; the renal secretion is often not at all increased, or only to an insignificant extent, because the more the venous blood is obstructed in the portal circulation, the less pressure is there in the arterial system; and this last circumstance has always an important influence over the secretion of urine. If diuretics are employed, we should at all events be cautious in prescribing Digitalis, Squill, and other remedies, which injure the stomach; and we should confine ourselves to such diuretics as Juniper Berries, Ononis Spinosa, Levisticum,[1] and bitter vegetable infusions. Saline diuretics, likewise, should not be given for any length of time.

The ascites is acted on more rapidly, and with greater certainty, by means of drastic purgatives, because the watery excretions from the intestinal mucous membrane operate directly upon the overflow of blood in the portal vein, and the intestinal secretion can be increased with far greater certainty than that of the kidneys. Colocynth, Gamboge, Elaterium, and similar preparations, however, very easily derange the digestion, and give rise to vomiting and a dangerous degree of collapse; moreover, it is often very difficult to check the diarrhœa. Uncontrollable purging is always to be dreaded when lardaceous infiltration of the liver coexists with the cirrhotic degeneration, because the intestinal mucous membrane is then often affected in a manner similar to the liver; it is also liable to occur when the hepatic affection is accompanied by advanced Bright's disease of the kidneys. When the ascites is so extensive that the respiration is impeded and the meteorism is very troublesome, the best plan is to remove the fluid by means of paracentesis. This operation is rarely dangerous from the supervention of peritonitis,[2] but must not be repeated unnecessarily, because the exhaustion is of course increased by the rapid return of the effusion and the remarkable loss of albuminous substances.[3]

As the disease advances, the main indications for treatment are always to assist, as far as possible, the nutrition of the body and the formation of blood, and to remove every cause of exhaustion. A diet, carefully selected in reference to its digestibility and nutritious value, is the first requisite; and in addition to this, provided there be no other indications of a more urgent character, we may try Extract of Cinchona Bark, Chalybeate Waters in small quantities, and other tonics.

The acute gastro-enteric catarrh, accompanied by typhoid symptoms,

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[1] The root of the *Ononis Spinosa*, one of the Leguminosæ, and the root of the *Ligusticum Levisticum*, belonging to the natural Order Umbelliferæ, are employed in Germany as diuretics.—TRANSL.

[2] In two cases only, have I seen paracentesis followed by inflammation of the peritoneum.

[3] It is obvious that the pressure of the ascitic fluid partially counterbalances that of the portal blood, and that thus the rapidity of the effusion diminishes with the increase of the abdominal dropsy. In performing paracentesis we give up this advantage; and hence this procedure should only be had recourse to as a matter of necessity.

which usually ushers in the last stage, may be treated by mineral acids, with the addition of Ether and other analeptics, or, when there is also profuse diarrhœa, by Calumba, Cascarilla, Tannin, &c.; but usually it is impossible to effect its removal or to defer the fatal termination. The same remark is equally applicable to the secondary pneumonia, pleurisy, peritonitis, and œdema of the lungs, for which the treatment appropriate to each must be employed.

Treatment by stimulants or narcotics is of equally little avail against the acholia, the advent of which is announced by the appearance of severe nervous symptoms. At this stage of the disease, all that remains to be done is to render death as easy as possible.

To the Cirrhotic Induration we have to add:

The simple Induration or Conversion of the Liver into Areolar Tissue.

Here a dense mass of areolar tissue becomes substituted for the parenchyma of the liver, from which, in many cases, every trace of the glandular tissue has disappeared over large spaces, whilst, at other parts, brown, uniformly-distributed dots of the remnants of the secreting cells can still be distinguished. The surface of the organ, under such circumstances, is sometimes smooth, and sometimes, on the other hand, covered with nodular eminences. The distribution of the vessels in the diseased parts is entirely changed; the mesh-like network of the branches of the portal vein has completely disappeared, and in its place we find elongated vessels, which can be injected both from the portal vein and the hepatic artery. It is only here and there, where there are still remains of the original tissue, that we observe the ordinary capillary distribution of the portal and hepatic veins. The extent to which the liver is involved in this induration varies; sometimes the induration extends throughout the entire thickness, while at other times it penetrates from the surface more or less deeply into the parenchyma, and is separated from the surrounding glandular tissue by a sharply-defined margin.

The simple induration may coexist with the granular (see Observation No. XXXV.); but, in other cases, the portions of the gland which are exempt, are perfectly normal. Of the larger vessels of the liver, I have in one case found the hepatic veins provided with valvular prominences, which narrowed the calibre of the vessel, and partially obliterated it. In this case old and recent extravasations of blood were disseminated through the parenchyma of the liver (Observation No. XXXVII.). The branches of the portal vein, however, and the bile-ducts permeating the indurated tissue, have not been constricted, but, on the contrary, dilated, in all the cases which I have examined. The causes of chronic inflammation of the liver, resulting in simple induration, are little known; in most cases we must attribute it to the same injurious agencies as operate in cirrhosis. In one of the cases which have come under my observation, the advent of the disease had been preceded by intemperate habits, in another by intermittent fever, and in two cases the chronic inflammation had extended to the hepatic tissue from the peritoneum.

The symptoms of simple induration agree in the main with those produced by granular induration. It is worth mentioning, however, that, according to my experience, the pain in the hepatic region which accompanies the origin of the induration, is usually greater and more extensive

than in cirrhosis; in one case the induration of the liver was preceded for many months by the symptoms of chronic peritonitis. The symptoms resulting from the obstructed circulation and excretion of bile are entirely the same as in cirrhosis; those referrible to the former cause were in one of my cases unusually well developed, owing to the circumstance that the intense inflammation of the capsule had produced a constriction, and, at some places, even an obliteration, of the hepatic veins.

An accurate diagnosis between the simple and the granular induration during life is in most cases impossible. It can only be arrived at when the situation of the liver and the nature of the abdominal walls permit of careful palpation. Practically, moreover, the differential diagnosis of these two conditions is of slight import, inasmuch as the prognosis and the treatment agree in all essential particulars. When the induration involves the greater portion of the gland, the prognosis is equally unfavorable as it is in the granular liver. In treatment there are no other remedies of service in the simple induration than those which are found useful in the granular liver. Permanent benefit can only be looked for where there is an early opportunity of treating continuously with general and local antiphlogistics, the inflammation which spreads from the capsule to the parenchyma; the symptoms of this inflammation are usually sufficiently marked.

Observation No. XXXVII.

Abdominal pain.—Circumscribed peritoneal exudation.—Slight Jaundice.—Improvement.—Six months afterwards, extensive Ascites.—Œdema of the lower half of the body.—Gangrenous Erysipelas.—Death.

Autopsy: Remains of old and recent Peritonitis.—Thickening of the Mesentery.—Numerous adhesions of the Spleen and Liver.—Hæmorrhage from the Stomach and Intestines.—A moderately enlarged Spleen.—Granular and Simple Induration of the Liver.—Constriction of the Hepatic Veins.

Maria Gittner, aged 38, a shoemaker's wife, was a patient in the clinical department of All Saints' Hospital from February 17th to March 2d, 1857.

Her disease commenced about Christmas, 1855, with pain in the abdomen and swelling in the left side of that region,—complaints which, with slight intermissions, had already lasted seven months, when the patient applied for advice at the Hospital on August 3d, 1856. At that time, she suffered from œdema of the feet and abdominal parietes and had slight jaundice. The abdomen was distended, and of a globular form, and yielded a clear tympanitic sound on percussion, except in the left hypochondrium, where there was an irregularly-defined dull space, the position of which was not altered when the patient lay on her right side, and around the margins of which the dulness gradually disappeared; this was regarded as a circumscribed peritoneal exudation—a supposition which was afterwards confirmed by *post-mortem* examination. The liver was of normal dimensions; the spleen could not be accurately defined, in consequence of the exudation. The digestive functions were undisturbed, except that the bowels were confined, and the stools rather light-colored. The urine was abundant, and free from albumen.

The patient was treated for chronic peritonitis; the pain ceased; the dulness on the left side was reduced, and the œdema diminished. All attempts, however, to remedy the patient's cachectic state were in vain, and she was discharged from the Hospital uncured.

On February 17th, she returned. The œdema of the lower extremities and of the abdominal parietes had again made their appearance, and had reached such an extent that there were moist excoriations upon the surface. The abdomen was distended by a large quantity of fluid and fluctuated; the diaphragm was pushed high up; great dyspnœa; sounds of heart normal and lungs healthy.

About eight quarts of clear yellow fluid were drawn off by paracentesis, whereupon the dyspnœa abated and the œdema diminished. Examination of the liver showed that it was slightly reduced in size, whilst granulations could be distinctly felt on its surface through the thin, flabby abdominal walls. The dulness had disappeared from the left hypochondrium; the spleen was enlarged, and its margins sharply defined. Urine reddish-brown, scanty, free from albumen and bile-pigment; appetite good; three thin motions of the bowels, containing but little bile, daily. The only diagnosis which could be formed was cirrhosis of the liver. Was ordered to have an easily-digested, nutritious diet, Red Wine and Decoction of Cascarilla.

February 25th, the diarrhœa had ceased; great tympanites; respirations slightly impeded; a moderate degree of jaundice; the water continued to flow through the punctured opening.

February 26th, livid erysipelas of the left thigh, rapidly passing into gangrene of the integuments; pulse 120; yellow-coated tongue; moderate thirst; one firm, brown stool; the jaundice diminished; urine free from pigment. Was ordered to take Muriatic Acid and Spirit of Nitric Ether.

February 27th, pulse 112; respirations 9; somnolence and typhoid delirium; one pale stool.

February 28th, pulse 120; respirations 10; the consciousness again clear; less jaundice; urine very scanty; tenderness of the hepatic region. The same treatment was continued.

March 1st, pulse 112; respirations 24; the gangrene of the thigh was extending in depth; the abdomen was again filled with fluid; the impulse of the heart could be felt in the third intercostal space. Urine very scanty; tongue dry; hiccough. Wine and Infusion of Valerian with Ether were prescribed.

March 2d, pulse 138; respirations 40; great collapse. Death occurred in the afternoon.

Autopsy, 16 *hours after death.*

The skin of the dead body was slightly jaundiced; the integuments of the lower half of the body were much swollen and œdematous; on the left thigh there was a superficial gangrenous ulcer.

The membranes of the brain were somewhat congested; the brain itself was normal.

The mucous membrane of the bronchi was injected and covered with bloody mucus; both lungs were firmly adherent; but the pulmonary tissue was healthy, with the exception of œdema and hypostatic congestion posteriorly.

The pericardium was united to the heart by numerous firm adhesions; the muscular tissue and valves of the heart were normal.

Many pounds of a yellowish fluid mixed with gray fibrinous flakes were found in the abdominal cavity. The peritoneum was opaque, at some parts injected, and at many places much thickened; many of the coils of intestine were adherent to one another.

The spleen was enlarged by about one-half: its capsule was thickened and callous, and connected to the surrounding parts by broad firm bands of areolar tissue, which stretched upwards towards the diaphragm and downwards to the sigmoid flexure,—in the latter locality enclosing yellow cheesy masses, the remains of the old circumscribed exudation. The mesentery and the mesocolon were considerably thickened.

The stomach contained a brownish-black bloody fluid; its mucous membrane was pale. The intestinal canal, throughout its entire extent, was filled with a similar bloody fluid; its mucous membrane was slightly injected and relaxed, and beyond the ileo-colic valve was bluish-black and œdematous.

The kidneys and urinary passages were normal. The uterus and ovaries were firmly adherent to the surrounding parts, but in other respects were unchanged.

The liver was reduced in size by about one-third. It was so intimately connected by firm areolar tissue to the diaphragm, the adjoining portions of intestine and the kidneys, that it was necessary to dissect it away with the knife. Its capsule was thickened and fibrous, and presented white coriaceous patches penetrating into the parenchyma; its surface was uneven and divided into numerous lobes of greater or less size. The terminations of the hepatic veins were closed by complete, or incomplete, partially perforated septa, and were surrounded externally by thickened sheaths of areolar tissue; the sheath of the portal vein and of the hepatic artery was likewise much thickened. From the outer surface masses of areolar tissue penetrated more or less deeply into the interior of the liver, destroying the glandular substance over large spaces, and only leaving the remains of it at isolated spots. Besides this, small and broad rims of areolar tissue were seen upon section, which were connected with the thickened sheath of the vessels and surrounded groups of hepatic lobules, partly healthy, but for the most part presenting a blue, brown, or dirty-red color from the presence of extravasated blood.

The gall-bladder contained a small quantity of turbid brown bile.

The ascitic fluid obtained by paracentesis was slightly turbid; after filtration, it deposited upon standing large flakes of fibrine, and subsequently a coagulum. It consisted of:—

Water	97·97
Solid constituents	2·03
	100·
Albumen	1·05
Extractive matter and salts	0·98
	2·03

It was found to contain small quantities of leucine, but no sugar.

The induration of the liver in this case was induced by an attack of peritonitis, the symptoms of which had preceded those of the hepatic affection by almost two years. From the capsule the inflammation penetrated into the interior of the gland, partly in a direct manner, and partly

along the sheaths of the vessels; it attacked the coats of the hepatic veins, and thus caused the formation of valvular occlusions in this vessel. To these occlusions must be attributed the remarkable obstruction of the circulation, which, independently of its effects upon the entire portal system, gave rise to extravasations of blood even in the hepatic parenchyma. It is worth mentioning, that, notwithstanding the remarkable hydrostatic pressure in the portal veins, the ascitic fluid did not contain a larger proportion of albumen than in other cases.

B. CIRCUMSCRIBED INFLAMMATION OF THE LIVER.

(*Hepatitis vera circumscripta, suppuratoria.*)

1. *Anatomical description.*

This form of inflammation of the hepatic tissue is always limited to one or several isolated patches; in these the process runs through its various stages, without the remaining portions of the gland being implicated to any great extent, with the exception of congestive turgor, which is rarely absent.[1]

As a rule, the inflamed patches are found in a condition of suppuration; one rarely has the opportunity of observing them at any other stage.

At the commencement of the process I have found the diseased portions of the liver partly of a red, and partly of a pale-yellow color. The redness disappeared at the margin, and merged into a broad yellowish rim. In one case I have noticed a branch of the portal vein filled with coagulated blood occupying the centre of the mass; the pale patches contained at some places accumulations of pigment of an ochre yellow color; the lobules were here remarkably enlarged and surrounded by pale-gray, translucent halos. The glandular tissue at the diseased portions is loosened, and is of softer consistence than natural, and in cases where the inflamed patches are at the surface they project beyond the normal contour of the gland in the form of flattened swellings, over which the capsule is opaque and injected.[2]

The suppuration usually makes its appearance at an early period. Isolated yellowish dots are first observed, which commence in the centre of the lobules, the margins still remaining firm; these gradually unite, forming small collections of pus, which increase rapidly, and, becoming incorporated with others in their neighborhood, at length give rise to extensive hepatic abscesses.[3]

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[1] Exceptional cases are met with, where the circumscribed inflammation occurs in conjunction with a diffuse form.

[2] According to Haspel, the first indications of inflammation of the liver are in the form of "de marbrures ecchymotiques, ou de tâches jaunâtres, laiteuses d'un rouge brun plus foncé que de coutume, noirâtre même, en quelques points."

[3] I have only been able to trace this process in the collections of pus which form in the liver in cases of pyæmia, and the above description applies to what I have observed in such cases. In other cases of hepatitis, the formation of pus does not necessarily correspond to the form of the lobules; cases, however, are met with in which the exudation process, although quite independent of phlebitis, assumes the precise lobular form. Annesley, Andral, Louis, and Stokes have already made similar observations; and Richard Quain (*Transactions of the Pathological Society of London*, Vol. IV.) describes with great minuteness a case of this nature, which, from its analogy to lobular pneumonia, he designated "lobular inflammation of the liver."

Whilst this process is going on, the hepatic cells are gradually destroyed; they become filled with a finely-granular albuminous matter, and by degrees are disintegrated, with the exception of the nucleus, which continues intact for a longer period.[1]

In recent abscesses the cavity is found filled with pale-yellow pus, and the walls consist of softened hepatic tissue, which hangs in shreds into the interior.

When the abscess has lasted for a long period, it undergoes various changes. In the first place, its walls become smooth, whilst the shreds of infiltrated hepatic tissue, which are bathed by the pus, are gradually destroyed and dissolved. The process of breaking down into pus then extends farther into the tissue infiltrated with exudation, and the abscess gradually assumes a rounded form, unless it becomes incorporated with other cavities in the immediate neighborhood, in which case a larger abscess is formed, with walls bulging in a sinuous manner, and traversed by cord-like processes or bridges of hepatic tissue.

In cases which terminate fatally at an early stage, the margins of the suppurating cavity either simply consist of softened, œdematous, hepatic tissue, or the cavity is lined by a thin, gray layer of fibrinous matter. On the other hand, when the abscess is of old date, a capsule of areolar tissue containing blood-vessels may be developed,[2] which limits the circumference of the mass, and at last causes it to be gradually absorbed, the walls of the abscess becoming approximated, and ultimately, under favorable circumstances, becoming united into a firm callous cicatrix. In most cases, a cheesy or calcareous remnant of the pus remains. A deep cicatrix-like depression of the glandular tissue is afterwards found in place of the cavity of the abscess.[3] The external cyst does not always limit the progress of the purulent destruction: in many cases it is broken through, and the remains of it are seen projecting into the collection of pus, whilst the inflammatory process extends beyond it. Very frequently it happens that no defined boundary is formed at all, but the inflammatory process continues to extend, until a perforation occurs, and the pus finds an outlet. The pus is rarely poured into the abdominal cavity, because at the places where there is danger of this occurring, adhesive in-

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[1] I once had an opportunity of tracing the changes which the hepatic cells undergo in a dried-up inflammatory deposit, the size of a hazel-nut.

[2] According to Haspel, the cyst may be completely developed at the end of from twenty to twenty-five days; as time passes on, it increases in firmness and thickness. Louis found this cyst composed of several laminæ, similar to those of a pleuritic membrane, and, at some places, of a cartilaginous consistence.

[3] Cambay (*loc. cit.*, p. 223) describes three cases of partly complete, partly incomplete, cicatrization of hepatic abscesses. In one case, all the symptoms of hepatitis disappeared. Two months afterwards, the patient died of tubercle in the lungs, and, on *post-mortem* examination, a depression, the size of a half-franc piece, was found upon the convex surface of the liver. Here the tissue was grayish-white and fibrous, and, on laying it open, a small deposit of pus, surrounded by condensed glandular tissue, was discovered.

In a second case, only one of the abscesses cicatrized, while the contents of the other were evacuated through the diaphragm, the pleura and the hepatized lung, into the bronchi.

In a third case, the occlusion of the suppurating cavity was incomplete; the small abscess was surrounded by radiating cicatrices, indicating the commencement of a curative process.

Haspel (*loc cit.*, pp. 239 and 240) observed numerous white, stellate, fibrous bands, indicating the remains of abscesses, the development and cure of which had, to some extent, been traced during life. Petit, in his Memoir on Abscess of the Liver, mentions one case, in which cicatrization took place after puncture of the abscess.

flammation of the capsule almost invariably occurs, and attachments are formed to the abdominal walls and the adjoining organs. When, however, the abscess does open into the abdominal cavity, the result is fatal peritonitis. The abscess frequently perforates the thoracic or abdominal wall superjacent to the liver, and opens directly outwards; or the pus, after burrowing downwards, may discharge itself into the pelvic, inguinal, or sacral region, or close to the spine, &c. Not unfrequently it takes an upward direction, penetrates the diaphragm, and empties itself into the right pleural cavity, or forces its way into the adherent right lung, inducing in this organ also a destructive separative process, or, in favorable cases, passing by a free opening into a bronchus, by which the pus is discharged externally.

The stomach, the duodenum, and the colon are the principal abdominal organs into which abscess of the liver discharges itself; it is only in rare cases that the pus is transmitted to the intestinal canal through the biliary passages, either by the gall-bladder or the smaller ducts.[1] To the exceptional cases belong such as have been recorded by Graves, Rokitansky, and Bentley,[1] where the abscess has found its way through the central portion of the diaphragm into the pericardium, and those rare cases, where it bursts into the portal vein or inferior vena cava.

Hepatic abscesses are sometimes superficial; at other times, and more frequently, they are deep-seated. They may be developed in any part of the gland, but most frequently they attack the posterior portion of the right lobe. Haspel found the comparative frequency of their occurrence as 1 in the left lobe to 30 in the right. In general, the deep-seated collections of pus are more tedious and dangerous than the superficial, which do not attain such a large size, and which are recognized at an earlier period by the occurrence of acute pain.

The size of hepatic abscesses varies greatly; not unfrequently they attain the size of a child's head, or are larger. Of the few cases which have come under my own observation, one measured 2, and another 5¾ inches in diameter; the most of those, which have been developed in consequence of pyæmia, have not reached the size of a hen's egg.[2]

The number of abscesses amounts in most cases to from 1 to 3, rarely to more; it is only the pyæmic deposits that are found in larger numbers, such as a dozen or upwards.

The purulent contents of the more recent abscesses are usually yellow, creamy, and destitute of odor; less frequently they are of a crumbly (*krümlich*) character, or mixed with blood or the remains of the disintegrated hepatic tissue. In old abscesses the pus sometimes emits a pungent ammoniacal odor, and occasionally it is mixed with bile of a yellow or greenish color, when, during the progress of the suppuration, any of the bile-ducts are opened and pour their contents into the abscess.[4] The cases where

[1] Cambay (*loc. cit.*, p. 534) found pus in the gall-bladder and the hepatic ducts communicating with abscesses. In another case, an abscess opened directly into the gall-bladder.

[2] *Transactions of the Pathological Society of London*, Vol. II.

Cases of hepatic abscess are met with of enormous size. Lieutaud, in one case, estimated the quantity of pus at twelve pounds. Annesley, in another, measured ninety ounces. Portal, Haspel, and other authors record cases where almost the entire liver was converted into an enormous cavity filled with pus.

[4] Rokitansky has found the bile-ducts terminating in the purulent deposit, by gaping, transversely- or obliquely-divided mouths; in exceptional cases, also, the sides of the ducts were laid bare and opened. Cambay has made similar observations (*loc. cit.*, p. 529).

the pus found in the liver appears reddish-brown, chocolate-colored, or like the lees of wine,[1] are much rarer than was formerly assumed, for it is only in exceptional instances that the blood-vessels are laid open during the progress of the suppuration.

The hepatic vessels are implicated in another way when the abscess approaches near to them; they inflame, and their lining membrane becomes rough and covered with fibrinous deposits, which fill up their calibre more or less completely. I have frequently observed this condition in the hepatic veins, and especially in conjunction with pyæmic abscesses of the liver. It is far rarer for the portal vein, isolated as it is by a thick sheath, to sympathize with the disease; but even cases of this nature have been met with. Russell[2] found a branch of the portal vein which was close to an abscess of the liver, in an inflamed condition; and, in the museum of Guy's Hospital in London, I saw the preparation of an abscess of the liver, which communicated with a thickened and inflamed branch of the portal vein.

It is far more common for the hepatic abscesses and the disease of the portal vein to stand in another etiological relation, the latter being the cause rather than the effect of the former, as is the case in the so-called pyæmic or metastatic abscesses of the liver. The development of these abscesses differs in many particulars from that of those which are the result of primary inflammation. In this case there are developed reddish-brown rounded deposits (*Heerde*), varying in size from a pea to a hen's egg, which are usually more numerous than in primary abscess. They exhibit a predilection for the periphery of the gland; they very rapidly pass into suppuration or an ichorous state, and rarely shrivel up and cicatrize. We shall have to consider these more in detail in treating of the diseases of the hepatic vessels.

The bile in suppurative hepatitis exhibits no change of any constancy; sometimes it is thin, at others thick and viscid; its color is brown, greenish, or reddish; it is only very rarely found to contain pus.

Circumscribed hepatitis is observed to terminate in induration and destruction of the diseased portions of the gland, as well as in suppuration. White fibrous callosities (*Schwielen*) of a radiating form are developed, which frequently enclose yellow cheesy masses. This form of inflammation has recently been described as syphilitic disease of the liver, and shall engage our attention particularly hereafter.

Termination in gangrene is extremely rare. The earlier physicians allowed themselves to be misled by the blackish discoloration of the circumference of hepatic abscesses, into the supposition that there was gangrenous disintegration, when there was really no gangrene present. True gangrene of the liver has been ascertained to exist in isolated cases by Andral, Rokitansky, Cambay, Haspel, Budd, and others. The cause of the gangrene has, in most cases, been the absorption of gangrenous matter into the blood, the deposits (*Heerde*) in the liver originating, like the similar affection of the lungs, from putrid infection in consequence of phlebitis. Thus Budd relates a case in which sphacelus of the toes had given rise to gangrenous patches in the liver, lungs, and spleen, together with suppuration in the hip-joint. In this case numerous small cavities were found filled with an ash-colored flaky mass. In Cambay's case there was a translucent bladder-like mass, 8 centimètres (3·15 Eng. inches) in

[1] Ἀμορχη of Hippocrates.

[2] Budd, *op. cit.*

diameter, in the right lobe of the liver, which, when opened, discharged a reddish fluid of a gangrenous odor. The wall of the cavity consisted of a soft, brownish-black, putrid substance, which could be washed away by a stream of water, when there was found beneath a grayish-yellow layer 1 centimètre (⅓ Eng. inch) in thickness, which separated the mass from the surrounding anæmic tissue of the liver. According to the experience of Cruveilhier, Haspel (*loc. cit.*, p. 165), and others, gangrene sometimes attacks the walls of an abscess after it has been opened and the air has been allowed freely to enter. Under certain circumstances, extreme exhaustion seems to induce gangrenous destruction of the walls of a hepatic abscess, as in the case of a man aged 60, recorded by Andral, who suffered from great inanition, in consequence of a large ulcer of the stomach.

The above are the principal morbid changes which the inflammatory process induces in the liver. In addition to them, there are usually anatomical lesions of other organs, which we must take into consideration in order perfectly to understand the pathological anatomy of the disease and obtain a clear insight into its nature. The most important and constant of these are found in the gastro-intestinal tract, the mucous membrane of which is usually the seat of exudation-processes and ulceration. In most cases these lesions are limited to the large intestine, and occasionally the lower portions of the ileum is also diseased, whilst in the upper portion of the small intestine and in the stomach, the only morbid appearances observed are slight hyperæmia and catarrh, and even these are by no means of frequent occurrence. The large intestine, however, in the majority of cases of abscess of the liver, presents important morbid alterations, especially in tropical countries; all gradations are met with here, from simple redness to brownish-black discoloration, and from œdematous thickening and slight superficial exudation to the most extensive ulcerations and gangrene. Of 29 cases of abscess of the liver collected by Annesley in the East Indies, in 21 there was dysenteric ulceration of the large intestine; of 25 cases which came under Haspel's observation in Algeria, 13 were of this nature; while of 17 cases observed by Budd, for the most part among sailors who had returned to England from hot climates, there was ulceration of the large intestine in 10.

In our own climate, the relation between the two affections is very different; hepatic abscesses are rarely attended by dysentery.[1] Of 16 observations collected by Louis and Andral, ulcers were present in only 3, and in 2 of these cases the ulcers were tubercular; of 8 cases, which have come under my own notice, there was intestinal affection in none.

A second class of lesions, with which abscesses of the liver are wont to be complicated, is inflammation of the veins and the numerous affections which give rise to this lesion and spring from it. In cold climates, these are the ordinary accompaniments of abscess of the liver, just as dysentery is in warm climates. The phlebitis may be seated in the roots of the portal vein, or in other veins, such as those of the extremities, the uterus, or the cranial cavity. Diseases of the excretory ducts of the liver, inflammation, dilatation, concretions, worms, &c., are complications of abscess of the liver, of still rarer occurrence than either the intestinal affection or phlebitis.

Of the thoracic organs, the right lung and the right pleura are often

[1] Cheyne (*Dublin Hospital Reports*, Vol. III.) has published observations in which they were.

found implicated by the pressure of the diaphragm upwards, by the spreading of the inflammation to the pleura, by perforation of the abscess, or lastly, by metastasis in consequence of consecutive *phlebitis hepatica.*

As a rule, no morbid changes can be discovered in the central organs of the nervous system or in the uro-poietic organs. The spleen likewise presents no uniform character; it may be normal, small, or even enlarged. With rare exceptions, Cambay found it small in abscesses of the liver accompanied by dysentery; Haspel, on the other hand, usually found it large, soft, and filled with dark blood. The mesenteric glands in most of the dysenteric cases are swollen, injected, soft, and rarely suppurating.

2. *Etiology.*

Inflammation of the liver terminating in abscess is a rare disease in temperate climates. According to the unanimous opinion of competent observers, it is much more frequently met with in warm climates and in the tropics, although even there the affection is by no means one of every-day occurrence.

It is not in every case that we succeed in discovering a cause for the inflammation. I have met with two cases of abscess of the liver of large size, without being able to determine with certainty the existence of any cause, even after the most careful investigation of the previous history.

The following we know to be causes:—

a. *Contusion of the Liver in consequence of a thrust, blow, fall, or other traumatic agency.*

Unequivocal cases of this nature have been mentioned by Andral, Budd, Morehead, and many authors; but they are not of such frequent occurrence as might have been expected. The violent contusions to which the liver is so frequently exposed by falls from a great height, or against sharp corners and edges, by severe blows, &c., on the whole, rarely give rise to traumatic hepatitis. I have had under my care a railway-laborer, whose right hypochondrium was crushed between the buffers of two railway-wagons, and who, in consequence of this, became jaundiced, but without any hepatitis resulting. It would thus appear that the liver has no great tendency to traumatic inflammation; and, moreover, the external violence must be particularly severe, or must co-operate with very unfavorable conditions when the ribs prove an insufficient protection and defence. Of 62 cases of hepatic abscess collected by Budd, there were only 2 in which the cause could be traced with certainty to any mechanical violence. Morehead, in his extensive experience (318 Observations), could only point to 4 cases of this nature.

b. *Metastatic or Pyæmic Inflammation of the Liver.*

This is of much more frequent occurrence than the traumatic form, from which it differs in many important particulars. The history of this form of inflammation of the liver is still obscure on many points, which require further elucidation.

The mode of origin of metastatic hepatitis from inflammation of the portal vein is simple and intelligible; but that observed in pyæmia, resulting from injury of other veins, is more obscure and difficult of explanation.

Cases of hepatic abscess arising from phlebitis of the portal vein are not unfrequently observed. Dance [1] met with suppurative hepatitis originating in this way on four occasions; once it supervened after cauterization of a cancer of the rectum, and in another case, after an operation for fistula in ano; in two other cases the primary cause was an operation for strangulated hernia, in which an irreducible portion of omentum suppurated.

Cruveilhier [2] has described the formation of hepatic abscesses, as supervening after violent reposition of a prolapsed anus. Jackson met with the same results in three cases at Calcutta, after the extirpation of hæmorrhoids. Buck has seen abscesses of the liver resulting from inflammation of the splenic vein. In treating of Phlebitis of the Portal Vein, we shall become acquainted with other cases of the same nature.[3]

The supervention of hepatic abscess from inflammation of the veins of the systemic circulation is of far more common occurrence. The earlier physicians regarded injuries of the head and the resulting phlebitis of the cavity and bones of the cranium, as peculiarly apt to give rise to it; but there is no close connection or sympathy between the head and the liver, in the sense assumed by Desault and Bichat. Abscess of the liver may follow phlebitis in the most varied regions of the body, in the upper as well as in the lower extremities; it may supervene upon the phlebitis resulting from venesection, wounds, fractures, &c., and likewise upon *phlebitis uterina*, &c.

It is difficult to show how in these cases the plugging (*Embolie*) of the hepatic vessels is produced. The structural elements of the blood, fragments of coagulum, plugs of pus, &c., contained in the veins of the general circulation, cannot reach the vessels leading to the liver, the hepatic artery, and portal vein, without previously passing through an interposed system of capillary blood-vessels. Hence we must assume that these bodies, after having already traversed the capillary system of the lungs, are arrested in the capillaries of the liver, either owing to the smaller size of these capillaries, or perhaps to the circumstance of the foreign bodies having increased in size during their sojourn in the blood; or we must have recourse to some other theory for the formation of the metastatic deposits in the liver. On carefully examining pyæmic abscesses of the liver we very frequently find the hepatic veins either completely or partially filled with coagula (*Thromben*): but I have never been able to discover this appearance in the hepatic artery or in the portal vein. These circumstances lend an air of probability to the view frequently expressed by Magendie, Meckel, and others, to the effect that the purulent deposits are the result of an occlusion of the hepatic veins, produced by the backward passage of thrombi from the vena cava. There is, however, no certain proof of the correctness of this opinion; my own observations are not in favor of it. It is true, that the penetration of masses of coagulum into the hepatic veins cannot be doubted; but it is improbable that these give

[1] Dance: *Archiv. Général de Méd.*, T. XIX., p 172.
[2] Cruveilhier: *Anat. Pathol.* Livr. XVI.
[3] The production of hepatic abscesses, in consequence of lesions of the portal vein, has frequently been traced experimentally by injection with quicksilver.

rise to the abscesses of the liver. After injecting metallic quicksilver into the jugular vein of dogs, I have repeatedly found globules of the metal in the hepatic veins, but never any deposits of pus in the liver. In one case the animal died twelve days after the injection, and abscesses were found disseminated through both lungs; the pleural cavities were filled with purulent exudation; a globule of quicksilver, almost as large as a pin's head, was found at three different places in the hepatic veins, and smaller globules were seen in the coronary veins; but the liver was free from all trace of inflammation and suppuration, and, even at the localities occupied by the globules of mercury, the walls of the hepatic veins exhibited no great morbid alteration. Thus, whilst suppuration had already taken place in the lungs, no traces of local inflammation could be discovered in the liver, although the foreign matter had been present in the hepatic veins for an equally long period as in the lungs.[1]

When, on the other hand, as has been shown by Cruveilhier,[2] the mercury is introduced into the mesenteric vein, or into other branches leading to the portal vein, numerous deposits of pus are formed in the liver in a short space of time. Disease of the hepatic veins cannot therefore be regarded as the cause of pyæmic abscesses of the liver; moreover, the mode of distribution of the inflammation in these vessels, and its occurrence at those places where the abscesses infringe laterally upon the wall of the veins, favor the idea of its being rather of a secondary character. The first supposition, according to which particles of coagulum, which have already passed through the capillaries of the lungs, are arrested in the capillaries of the hepatic artery, is therefore more probable. There is no certain proof, however, of the correctness of this view.

Purulent deposits in the liver supervening upon endocarditis are extremely rare, if they ever occur; I have never myself met with an unequivocal case of this nature. Virchow[3] records one observation, showing that deposits of pus may be developed in the liver through the medium of the hepatic artery. In an individual suffering from gangrenous infarctions of the lungs with hæmoptysis, an ichorous discharge was taken up into the pulmonary veins, and, being borne along in the circulation, gave rise to metastatic deposits of pus in the heart, brain, spleen, liver, kidneys, and skin, and also to obstruction of the mesenteric artery.

It follows that if we adhere to the strictly mechanical theory of their formation (which, however, is not by any means to be regarded as settled), the mode of origin of pyæmic deposits in the liver is, as yet, far from satisfactorily explained.

c. *Inflammatory and Ulcerative Processes in the Gastro-intestinal Canal.*

The frequent coexistence of diseases of the intestine with inflammation of the liver, already mentioned, for a long time led to the opinion that there was an intimate etiological relation (*Causalnexus*) between the two, although it was not decided what was the real nature of this relation. Broussais regarded inflammation of the intestinal canal as one of the most

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[1] Gaspard (*Journ. de Physiol.*, T. I., p. 165) found the globules of mercury in one case surrounded by small abscesses of the liver.

[2] *Anat. patholog.* Livr. XI.

[3] Virchow: *Archiv für Patholog. Anatomie.* Bd. I., s. 332.

common causes of inflammation of the liver, and in his 149th Proposition he laid down the rule: "L'hépatite est consécutive à la gastro-enterite, quand elle ne dépend pas d'une violence extérieure." According to him, the irritation was transmitted from the mucous membrane of the intestine, but particularly from the duodenum, along the bile-ducts to the parenchyma of the liver. Andral, and likewise many other French physicians, adopted the same explanation, not merely for acute hepatitis, but also for the chronic form terminating in induration:—"Dans plus d'un cas l'observation des symptômes conduit à partager l'opinion de M. Broussais, qui admit, que dans la plupart des cas de phlegmasie il y a en d'abord duodénité."[1] In Germany and England, however, this view has met with less support, and deservedly so. It is true that inflammations of the lining membrane of the intestine are propagated along the bile-ducts, and that jaundice not unfrequently arises in this way; but in most cases of hepatic abscess we are quite unable to trace any such anatomical continuity of propagation, while, on such a supposition, hepatitis ought to be infinitely more frequent than it really is.

According to another view, first brought forward by Ribes, the inflammation is propagated from the gastro-intestinal mucous membrane to the liver, through the medium of the veins. Latterly it has been the custom to explain in this way the coexistence of abscesses in the liver with tropical dysentery, the origin of the former being referred to phlebitis of the roots of the portal vein. Budd adopts this theory in a still wider sense, inasmuch as he endeavors to explain by it, not merely the abscesses of the liver which accompany dysentery, but also those which are found in conjunction with inflammation and ulceration of the bile-ducts, simple and cancerous ulcers of the stomach, and softening of the gastro-intestinal mucous membrane. In addition to the inflammatory products, which become mixed with the blood of the portal vein, in consequence of inflammation of the small veins, the blood, according to Budd's idea, may be contaminated by the absorption of other substances resulting from softening of the tissues, and also by the imbibition of fœtid gases and fluids from an intestine affected with dysentery, so as to give rise to hepatitis. The absorption of the products of inflammation into the veins is thought to produce numerous small collections of pus; whilst, on the other hand, the absorption of other substances gives rise to diffuse inflammation and single large abscesses. The latter portion of the theory can neither be confirmed nor refuted; it is possible, yea probable, that deleterious matters are absorbed; but as yet this has not been proved, and at the same time we are equally unable to explain why the absorption of such matters does not occur under other favorable circumstances, as in typhoid and tubercular ulceration of the intestines, in the diarrhœa of temperate climates, &c. We are likewise ignorant as to the manner in which the portal blood, thus contaminated, induces diffuse hepatitis, or indeed whether it does so at all.

Meanwhile, even the first portion of the theory is by no means sufficiently established, viz., that, in cases of dysentery and of ulcers of the stomach, and intestinal mucous membrane, it is phlebitis of the roots of the portal vein in the diseased portions of intestine that produces abscess of the liver. It has been far too generally assumed that the dysentery precedes the hepatic inflammation; almost all physicians who have acquired their experience in warm climates have arrived at the conviction that

[1] Andral: Clinique Médic., T. II., p. 289 and p. 439.

the dysentery is often preceded by the hepatitis, or that the two may be developed simultaneously, and that hepatitis frequently occurs without any disease of the intestine.[1]

These remarks are still more applicable to the hepatic inflammations of our own climate; a careful analysis of cases of this nature is entirely opposed to the idea of their connection with intestinal ulceration.[2]

Moreover, as regards the cases where the dysenteric ulceration of the intestine has preceded the development of the abscesses of the liver,[3] it has not as yet been proved that the hepatic disease is the result of the intestinal affection, and, above all, no one has succeeded in demonstrating, as the connecting link, any implication of the roots of the portal vein in the intestinal inflammation, or an intestinal pylephlebitis.[4] Cambay never found either the mesenteric veins or the portal vein inflamed; out of 4 cases, Mühlig[5] only once observed small yellow coagula, which appeared to him to be half disintegrated, in the superior mesenteric vein. Although it may easily happen that diseases of the fine venous twigs in the intestines may be overlooked, still there can be no doubt that a positive proof is possible, and that a positive proof must be given, before this

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[1] Cambay (*loc. cit.*, p. 212) observes: "Nous croyons en effet que l'on a trop généralisé, lorsqu'on a avancé qu'en Algérie la dyssenterie ou la diarrhée précédaient presque toujours l'hépatite, parceque nous avons observé plus de cas d'hépatite idiopathique non précédés du flux abdominal, que de ceux qui en etaient précédés."

Annesley also states that in the East Indies the hepatitis usually precedes the dysentery. More recently similar observations have been made by Morehead. This author has described 17 cases of abscess of the liver without any implication of the intestine, and 7 where the dysentery was secondary to the hepatic affection.

(Waring states, that of 300 cases of hepatic abscess proving fatal in the East Indies, in only 82 cases, or 27·3 per cent., was the hepatitis preceded by symptoms of dysentery, while in 204 of the cases where the condition of the large intestine was noted after death, there were no ulcerations, cicatrices, or abrasions in 51, or in exactly one-fourth.—*An Enquiry into the Statistics and Pathology of Abscess in the Liver.* By Edward John Waring. Trevandrum, 1854.)—TRANSL.

[2] Bristowe (*Transactions of the Pathological Society*, Vol. IX.) has carefully analyzed the observations bearing upon this point, collected in St. Thomas's Hospital at London. Of 324 cases of intestinal ulceration, 167 were tubercular, and in 12 of these cases small tubercular cavities were found in the liver. In 25 cases there were malignant ulcers of the stomach or intestine, and in 45 cases typhoid ulcers; in 55 cases the ulcers presented a simple character, and in 32 that of dysentery. Abscesses of the liver were only observed in 4 of the last-mentioned cases. In 3 of these cases, moreover, the dysentery was secondary, and in one case only was there an obscure connection between the abscess of the liver and the ulceration of the bowel obscure. Of 31 cases of abscess of the liver 12 were simple tubercular deposits, 5 were due to general pyæmia, and 4 were produced by hydatids; 10 were examples of the so-called idiopathic abscesses, and of these 6 were associated with ulceration of the intestines. The intestinal ulceration was twice produced by the evacuation of the contents of the abscess into the bowel; the 4 remaining cases were all that could be regarded as examples of the dependence of hepatic disease upon dysentery.

[3] According to Cambay, only 1 out of 20 cases of dysentery in Oran is complicated with hepatitis.

[4] Andral (*loc cit.*, p. 290) records two observations, which he believes, although scarcely with justice, may be regarded as proofs of a propagation of the inflammation from the mucous membrane of the bowel to the liver. In one case, in addition to great vascular injection of the ileum, the lining membrane of the inferior mesenteric and portal veins was reddened, whilst the splenic vein appeared pale. Along with this, the liver was large and congested. In the second case the liver was indurated, the lining membrane of the portal vein was red and easily torn, while the inner surface of some of its branches was covered with a sort of pseudo-membrane. The lining membrane of all the other vessels was pale. The mucous membrane of the stomach and intestines was in a state of chronic inflammation.

[5] Mühlig: *Zeitschrift der Gesellsch. Wiener Aerzte.* Bd. VIII., s. 1 u. 3.

view can be looked upon as established. This is the more necessary, inasmuch as the abscesses differ from those of pyæmia in their general characters, their limited number, &c., and also because the typhoid symptoms of pyæmic intoxication are absent in hepatitis accompanied by dysentery, and because enlargement of the spleen, according to the observations of Cambay, does not occur in the latter form of the disease. Hence the connection between intestinal ulceration and hepatitis, as cause and effect, has been by no means established, although it cannot be denied, that, in exceptional cases, under favorable circumstances, dysenteric and other forms of ulcerations of the bowel may possibly induce phlebitis of the roots of the portal veins, and so give rise to abscess of the liver. It follows, that we are far from participating in the opinion of Budd, according to whom almost all cases of hepatic abscess, which are not due to external violence, must be referred to purulent infection of the roots of the portal vein, resulting from ulceration of the mucous membrane of the stomach, intestines, or bile-ducts.

The idea of Morehead, who makes the cachexia induced by residence in the tropics the cause of hepatic disease, and who attributes the immediate exciting cause to a chill, is the view which is most in harmony with the facts, in the case of those abscesses of the liver which accompany tropical dysentery.[1]

Annesley, as the result of his extensive experience in the East Indies, was led to regard the relation subsisting between diarrhœa and abscesses of the liver, as entirely different from that above mentioned. For the cases, where the hepatitis preceded the intestinal affection, he assumed the existence of a reversed relation between the two, referring the inflammation of the bowel to a morbid quality of bile secreted by the diseased liver. The natural objection, that the small intestine, with which the bile first comes into contact, remained exempt, whilst it was only in the large intestine that the inflammatory process attained a marked development, he attempted to explain away by the circumstance of the longer delay of the fæces in the colon, and by the greater tendency of this portion of the bowel to exudation processes. Acrid decomposing fæcal matter, or indurated masses of fæces have frequently been thought to exercise an influence in giving rise to diarrhœa, while, in the case under consideration, the circumstance of the prominent folds of the bowel, which are most exposed to the contact of the intestinal contents, being the parts which are first diseased, favors Annesley's view of the matter; but, as yet, inflammation of the liver has not been shown to give rise to any change in the bile, of such a nature as to lend any support to such an opinion.[2]

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[1] Rouis (*Recherches sur les Suppurations Endémiques du Foie.* Paris, 1860) expresses his opinion as to the mode of origin of hepatic abscesses in hot climates, as follows :— "En résumé, l'irritation, qui envahit, le foie durant la saison des chaleurs, nous paraît dépendre de ce que cet organe est celui auquel le sang, sous l'influence de la raréfaction imprimée à l'air par le haut degré de la température, apporte le moins d'éléments gazeux." The further proof of this theory is, however, purely hypothetical.

[2] Rouis (*loc. cit.*, p. 224) coincides with this view in the main. He observes: "La production des accidents intestinaux semble donc se rattacher à ce que, la bile est versée en quantité trop grande dans les portions extrêmes du tube digestif, lesquelles, d'ailleurs se trouvant déjà congestionnées et irritées par le gêne, qu' éprouve leur circulation veineuse, doivent être d'autant moins aptes à supporter l'impression de ce fluide."

d. *Inflammation and Ulceration of the Bile-ducts.*

Inflammation and ulceration of the bile-ducts, and particularly that form which is induced and kept up by the presence of concretions, or, still more rarely, by the entrance of round worms, may give rise to hepatitis and abscess, owing to the inflammatory process implicating the parenchyma of the liver. Abercrombie long ago published two observations (Observations No. CXXVIII. and CXXIX.), where abscesses of the liver coexisted with gall-stones and great irritation of the bile-ducts. Bright (*Guy's Hosp. Reports*, Vol. I., p. 630), found numerous deposits of pus in the liver in a case, where there were gall-stones and ulceration of the gall-bladder. Louis (Observation No. V.) has described a case where the liver contained from thirty to forty abscesses, each the size of a pea; all the other organs were normal, except that the gall-bladder contained several calculi, which had given rise to ulceration of the walls of the gall-bladder, partly superficial, and partly deep.

Budd, who has published similar observations, refers the abscesses (*loc. cit.*, p. 92), like those which occur in dysentery, to the ulcers of the mucous membrane of the biliary passages, the venous twigs opening into the portal vein being the medium of communication. In the cases, which I have had an opportunity of observing myself, such a complicated mode of explanation was unnecessary, inasmuch as the bile-ducts were at some places destroyed by ulceration and the inflammatory process had implicated directly the adjacent parenchyma of the liver.

The abscesses, which have frequently been observed to arise from the penetration of intestinal worms (*Ascaris lumbricoides*) into the biliary passages, are produced in a similar manner. These worms produce at one time catarrh with uniform or saccular dilatation of the ducts, and, at another, erosion of the ducts and abscesses. The abscesses are usually multiple, and may, or may not, be in direct connection with the ducts. Not unfrequently they attain a considerable size, and open in various directions. Kirkland (*An Enquiry into the present state of Medical Surgery*, London, 1786,) observed an intestinal worm escape with the pus from a hepatic abscess, which had opened internally. Lobstein (*Journ. Complément.*, Tom. XXXIV., p. 271) and Lebert (*Traité d'Anatom. Pathol.*, Paris, Tom. I., p. 412) have described cases where the abscess perforated the diaphragm and reached the lungs. In addition to the above instances, Tonnelé (*Journ. Hebdom.*, Paris, 1829, Tom. IV.) and Forget (*Union Médic.*, 29 Mai, 1856) have observed multiple, hepatic abscesses produced by intestinal worms. The reader is also referred to the remarks to be made hereafter under the head of "Entozoa of the Liver," showing that abscesses may result from suppuration of hydatid cysts.

Besides the causes of hepatitis which have just been enumerated, there are others whose mode of operation is still less clearly demonstrable. To this class belong the influence exerted by hot climates and marsh miasmata, spirituous drinks, spices and other acrid articles of diet, chills, &c. The importance of these causes has already been considered in the chapter on Hyperæmia. (See pp. 42 and 46.) It is difficult to determine whether these causes are of themselves sufficient to give rise to abscess of the liver. At all events, there can be no doubt that cases of inflammation of the liver occur not unfrequently, the real cause of which cannot be discovered. This does not appear strange, when it is remembered that

the mode of origin of other inflammations, which are of more frequent occurrence and more accessible to observation, such as pneumonia, &c., is equally obscure.

3. *Clinical History and Symptoms.*

It is extremely difficult to lay down a clinical history of hepatitis which would be universally applicable, inasmuch as the complex train of symptoms presents an endless variation, depending upon the site of the inflammatory deposits and the concomitant morbid processes. In the first place, there are cases where the existence of inflammation is not indicated during life by any local change or functional derangement, and yet, upon *post-mortem* examination, abscesses of the liver, of a remarkable size, have been found quite unexpectedly. Andral (*loc cit.*, Tom. II. p. 303) gives the history of a young man, who, after a fatiguing ride on horseback, had an attack of fever, for which no local cause could be discovered. On the fourth day, he had a rigor and severe headache, his tongue was white, he had no appetite, and suffered from obstinate constipation; on the twelfth day, he began to be delirious, and on the seventeenth day he died. During the entire progress of the disease, the epigastrium and right hypochondrium were free from pain and tension; there was no vomiting and no jaundice; the various internal organs were carefully examined, without discovering any local disease. At the autopsy, all the organs were found free from any lesion of importance, and even the liver appeared normal, until an accidental puncture with the scalpel laid open an abscess, as large as an orange, filled with yellowish-white pus, destitute of odor, and surrounded by softened, red, hepatic tissue. Abercrombie, Annesley, Cambay, Haspel, Budd, and others have met with similar cases. Budd (*Diseases of the Liver*, 3rd Ed., p. 108) records the history of a Lascar, 62 years of age, who was admitted into the Seaman's Hospital with general emphysema and catarrh. The patient suffered from hectic fever, which led to the suspicion that he had tubercle. He grew weaker and died, without ever complaining of pain or tenderness in the right hypochondrium, and without ever having had vomiting, diarrhœa, or jaundice, or any symptom to indicate disease of the liver. On examination, an encysted abscess, containing more than a pint of pus, was found in the substance of the liver. The stomach and small intestines were normal, but the lungs were extremely emphysematous and the bronchial tubes filled with mucus. In another case, communicated by Dr Inman, of Liverpool, there was protracted diarrhœa with great emaciation, but no abdominal tenderness. At the autopsy, the large intestines were found extensively ulcerated, and there were three abscesses of the liver, containing in all about twenty ounces of pure yellow pus. (Budd, *loc. cit.*, p. 109.)

Two cases of large abscesses of the liver, the existence of which was not indicated during life by any local symptom, have come under my own observation. One was that of a man aged 34, who had been treated for chronic nephritis and exudation into the left pleural cavity. In addition to the morbid changes in the kidneys and pleura, an abscess five-and-a-quarter inches in diameter, with a dense capsule of connective tissue, was found in the right lobe of the liver, which, by the way, was not enlarged to any great extent; there was likewise another smaller abscess, the size of a hazel-nut, in the lobus Spigelii. Besides these two abscesses, there was a purulent deposit, the size of a walnut, in the abdominal muscles, in the

left iliac region. Nothing could be made out in reference to the etiology of the case. The second case occurred in an old man, who had been under treatment in the Hospital for a short time only, and who had formerly led a vagabond life. He stated that he had suffered from intermittent fever, and was admitted in a state of great prostration, with œdema of the feet, ascites, a pale, waxy-yellow color of the skin and other indications of cachexia. The spleen and liver were carefully examined, but no swelling or tenderness could be discovered. The power of digestion was completely destroyed; there was vomiting of gray, or sometimes of yellowish mucus; the bowels were constipated and the stools were normal in color. At the autopsy, in addition to chronic catarrh of the stomach, pulmonary emphysema and extensive atheromatous disease of the blood-vessels, a cavity, the size of a goose-egg filled with greenish-yellow pus, and with smooth, dense walls, indicative of a lengthened duration, was found in the right lobe of the liver, near its rounded margin.

In other cases, the symptoms of morbid processes consecutive upon the hepatitis, or which precede and give rise to it, are the most prominent, whilst the essential, fundamental lesion, is indicated either by no symptoms whatever, or by insignificant derangements, which are easily overlooked. In our own climate, this is in a marked degree the clinical history of the so-called pyæmic infection, in the course of which latent hepatic abscesses are wont to be developed. In warm countries, it is mostly dysentery or intermittent fever, and, according to Haspel, particularly the tertian form, which masks inflammation of the liver. Sometimes morbid processes induced by the hepatitis, such as peritonitis, or, still more frequently, pleurisy or right pneumonia, give rise to such prominent symptoms, that the real seat of the disease is overlooked and mistaken.

Lastly, cases occur in which hepatitis is developed under the mask of non-febrile gastric catarrh, and the existence of a more important lesion is only indicated at an advanced stage of the disease, by the occurrence of irregular attacks of rigors.

More frequently than these last-mentioned latent forms, we meet with other cases, in which the inflammation of the liver is characterized by a series of definite symptoms, which direct attention to the seat of the disease from the beginning, and which enable us to trace its various modes of termination. These local symptoms, however, are often so ill-defined, or so obscured by others, that it is a difficult matter to analyze correctly the manifold varieties which they present, according to the stage of the disease and other circumstances, to distinguish between the essential and non-essential symptoms, and to separate the primary and consecutive derangements from those directly referrible to the fundamental lesion. In a very large number of instances, the diagnosis does not rest upon that infallible basis, upon which we are enabled to build our conclusions in the case of diseases of other organs.

In giving a summary clinical history of the disease, it is necessary to select for description simple cases, having a traumatic origin, because in these cases complications are of least frequent occurrence.

After a thrust, a blow, or a fall against the hepatic region, the patients complain of pain and tenderness of the right side; the hypochondrium feels hard and tense, and, in most cases, an enlargement of the liver can be made out by means of palpation or percussion. This enlargement at one time extends upwards into the thorax; while, at another, the inferior edge is found at an unusually low level below the margin of

the ribs. The enlargement is accompanied in many cases by jaundice; but more frequently the color of the skin remains unchanged. The swelling and tenderness of the liver are usually attended by more or less severe febrile symptoms and derangements of the stomach. The skin is hot and dry; the pulse increases in frequency; the tongue is covered with a gray or yellow coat; not unfrequently there is bilious vomiting; the bowels are sometimes confined, and at other times relaxed, with bilious evacuations. When the pus is deposited near the upper part of the organ, the respiration is interfered with; the action of the right half of the diaphragm is impeded; the hepatic dulness extends higher into the thoracic cavity; a short, dry cough sets in, and sometimes pains are complained of in the right shoulder.

As soon as suppuration commences, the gastric disturbances usually become aggravated; the fever increases in intensity, and the patient is attacked at irregular intervals by rigors, followed by heat and profuse, exhausting sweats. When the abscess is completely developed, the general enlargement of the liver usually diminishes, and, in cases where the situation of the abscess is favorable, a circumscribed, fluctuating tumor may be felt. In many cases, the abscess remains surrounded by the hepatic parenchyma, and its detection is impossible.

The further progress of the disease varies greatly in its character. Under favorable circumstances, the suppurative process is arrested, the pus undergoes a retrograde metamorphosis, the abscess is gradually reduced in size and cicatrizes, whilst the symptoms, one after another, disappear. When the suppuration advances, and the abscess is not circumscribed, death usually ensues under typhoid symptoms, or those of hectic fever leading to exhaustion. In other cases, peritonitis sets in, which at first is of a local nature, but soon extends over the entire peritoneum and proves fatal.

When the abscess makes its way outwards, or into any of the neighboring organs or cavities, the symptoms vary extremely according to the direction taken by the pus. When the abscess bursts into the peritoneum, the result is peritonitis, speedily proving fatal. When it takes an outward direction, a fluctuating tumor filled with pus is developed in the hepatic region, or, owing to the contents of the abscess burrowing downwards, in the dorsal or pelvic regions. When the abscess opens into the stomach, there is vomiting of pus; when it opens into the intestinal canal, or, as rarely happens, into the bile-ducts, pus is voided with the stools. When the abscess penetrates into the cavity of the right pleura, it gives rise to the ordinary train of symptoms characteristic of pleuritic exudation. When it finds its way into the adherent right lung, suppurative pneumonia ensues, and when a communication is established between the purulent deposit and the bronchi, pus of a bloody, putrid character, is usually expectorated.

The above general sketch of the simplest form of hepatitis is but rarely applicable to the cases met with in practice, inasmuch as they are usually of a more or less complicated character. In order to become acquainted with these cases, it is necessary to subject the different symptoms to a careful analysis, in respect to their origin and importance.

Special Symptomatology.

a. *Local Symptoms.*

1. There are cases, where simple inspection furnishes sufficient data for diagnosis. At one time, the region corresponding to the contour of the liver is uniformly more bulging than natural; while, at another, and particularly when the abscess takes the direction of the abdominal walls, there is at one place a rather flattened prominence. Usually, however, the right hypochondrium is examined in vain for any obvious alteration, and hence directions are given to determine accurately the size and form of the organ, by means of percussion and palpation. Manipulation in many cases occasions increased tenderness, extending over the entire gland, or limited to one spot.[1] In cases where the situation of the purulent deposit is favorable, the diseased portion may be felt prominent and dense, or, when suppuration has already commenced, fluctuating. When, on the other hand, the deposit has its seat in the convex portion of the gland, as is so frequently the case, the form of the lower margin remains unaltered, and then, although the abscess may occasion fluctuation of the intercostal spaces, it is to percussion that we must look for assistance. Under such circumstances, the margins of the liver ought to be determined in every direction by means of the pleximeter, because the enlargement is rarely of a uniform character, and, as a rule, semi-globular prominences stretch upwards into the thorax or in some other direction, which might be easily overlooked were the diameter of the organ determined at one place only.[2] It must not, however, be forgotten, that cases are not unfrequent, where local examination furnishes no data whatever for forming a diagnosis, where neither the size nor the form of the gland is altered, and where there is no increase of tenderness. Andral, Catteloup and others, have recorded observations of this nature, and such cases have repeatedly come under my own notice.[3]

2. The pain met with in hepatitis varies greatly in its nature and severity; in many cases, it is entirely absent, particularly when the inflammation is deep-seated in the parenchyma of the gland, while, on the other hand, it is acute, when the inflammation approaches the outer surface, or when the serous covering of the gland is involved. Out of 177 patients, Rouis ascertained that 141, or 85 per cent., presented this symptom. The pain rarely exists from the first; usually it does not begin until suppuration has commenced, and terminates when this has ceased. The situation of the pain depends upon that of the disease; at one time it is in the right hypochondrium, at another, in the epigastrium; at another, it is situated posteriorly at the base of the thorax, or wherever else the morbid

[1] It is necessary to bear in mind the tension of the abdominal muscles produced by palpation, and particularly of the rectus muscle, which Twining regarded as an important indication of deep-seated hepatic abscess.

[2] The reader is referred to a case of abscess of the liver produced by the suppuration of a colony of echinococci, to be subsequently recorded.

[3] According to the valuable and carefully-collected observations of Rouis (*op. ant. cit.*, p. 115), an increase in the volume of the inflamed liver was observed in 73 out of 122 patients, or in 60 per cent. Taking the results of *post-mortem* examinations, the proportion was somewhat larger, or the liver was enlarged in 70 out of 101 cases. In most cases, the increased volume was first observed at the commencement of the suppuration; in a few cases only did it precede, or appear simultaneously with, the other symptoms.

process is developed; it is usually local and circumscribed, but often widely extended. In most cases, it is at first of a dull, tight character, and only becomes pricking or throbbing, with the commencement of suppuration; it is aggravated by percussion, and not unfrequently it is first excited in this way. In addition to the local pain, there exists in many cases (according to the experience of Rouis, in 28 out of 163 cases, or in 17 per cent.), a sympathetic pain, usually having its seat in the right shoulder, but sometimes complained of in the scapula and arm; the sensation is that of tightness or tension, or sometimes of an intolerable boring, and it is aggravated by every concussion of the hepatic region. This symptom usually lasts for a few days only, and ceases as soon as the pus finds an outlet. In one case, Rouis observed that the deltoid muscle became atrophied after a time. Rouis doubted whether this sympathetic pain were a symptom of hepatitis, because he failed to meet with it in any of his five Observations. Budd found it five times in 15 cases. Annesley believed, that the pain in the shoulder indicated with certainty, that the convex portion of the right lobe was diseased.

3. Jaundice is rather a rare symptom in hepatitis. Cassimir Broussais observed it 23 times in 66 cases, and Rouis 26 times in 155 cases. It is usually slight and of short duration, commencing in most cases at the time of the formation of the pus, rarely earlier, and sometimes, not until a short time before death. Thus jaundice is a symptom of no value in the diagnosis of suppurative hepatitis; its development may be accounted for by compression of the large bile-ducts by the inflammatory deposit,[1] or partly by the coexistence of catarrh of the ducts.

b. *Derangement of the Digestive Organs.*

During an attack of hepatitis, the functions of the digestive organs usually remain perfectly undisturbed, and it is only at the commencement of the suppuration that the tongue becomes covered with a gray or yellowish coat. Of 143 cases of acute hepatitis observed by Rouis in Algeria, there were only 30, or 21 per cent., where the hepatitis was accompanied by symptoms of gastro-enteric catarrh, loss of appetite, nausea, pain, and tension of the epigastrium, vomiting, semi-fluid bilious stools, &c. These symptoms, in most cases, lasted four or five days. Severe, persistent vomiting occurred when the stomach was compressed by a projecting abscess, and was then a symptom of ominous import, as regards prognosis.

Annesley imagined that the projection of red papillæ through a gray coating of the tongue, which afterwards became brown or brick-red, was a valuable indication of commencing acute inflammation of the liver; but the observations of French physicians, and particularly of Haspel, have not confirmed this opinion. The existence of a critical salivation, which was thought by Portal, Wedekind, P. Frank and others, to accompany hepatitis, has likewise not been confirmed.

In the chronic form of hepatitis, the gastric derangements are somewhat more frequent in their occurrence; but are usually less marked and only of transient duration.

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[1] Cruveilhier (*Diction. de Méd. et Chir. pratiq.*) succeeded in all such cases in discovering a mechanical obstruction to the excretion of bile; whereas Rouis maintains that the existence of such an obstruction is uncertain.

The frequency of diarrhœa, and particularly of dysentery, as accompaniments of the hepatic disease, has already been considered under the head of Etiology.

Peritonitis and ascites constitute other derangements of the abdominal organs, met with in hepatitis. The former makes its appearance with tolerable frequency, and is either local and circumscribed by adhesions, or general; in the latter case it sometimes is developed as *peritonitis ex perforatione*, in consequence of the bursting of an abscess into the abdominal cavity, and proves rapidly fatal. Ascites is of much rarer occurrence, and is produced by closure of the portal vein, in consequence of the compression of this vessel by the inflammatory deposit. Haspel observed two cases of this nature.

c. *Derangements of the Respiration.*

Various derangements of the respiratory process manifest themselves in the course of hepatitis, which sometimes are dependent upon the acute pains in the liver, at others upon the increased volume of the organ, or upon the propagation of the inflammation to the pleura or lungs, or upon the entrance of pus into these parts.

If, in consequence of the pain, the action of the diaphragm be impeded, the respiratory movements become thoracic, short, frequent, and, as it were, arrested. Sometimes a cough is also developed, which was spoken of by Hippocrates as "Tussis arida, sicca, molesta quidem, sed rara." The derangements produced by the enlargement of the gland are usually of a slighter nature, and only give rise to great dyspnœa when the patient moves. In such a case, the lower margin of the displaced lung can be made out by means of percussion; faint, or indistinct vesicular breathing, or sometimes bronchial breathing, is audible over the compressed portions of lung.

When the inflammation spreads from the liver to the pleura, or when the abscess bursts into the pleural cavity, the symptoms of acute or sub-acute pleurisy manifest themselves, the existence of which can be easily ascertained with a little care.

In cases where the inflammation is propagated from the liver to the lung, a sub-acute infiltration of the right lower lobe is developed, and is accompanied by the usual signs of pneumonia, until after the bursting of the abscess, when a large quantity of pus is expectorated. The subjective symptoms all this time are often insignificant; the cough occurs only in paroxysms, whilst in other respects the breathing is quiet, or there is only dyspnœa upon exertion. To this, however, there are exceptions, where the patients are tormented by severe, almost uninterrupted attacks of asthma. This is particularly liable to happen when the diaphragm contracts numerous adhesions, when its muscular tissue is destroyed, when there is extensive hepatization, or when large cavities are developed in the lungs.

It need scarcely be mentioned, that the respiration often remains perfectly normal in hepatitis, especially when the deposit is deep-seated in the gland, and when neither the diaphragm nor the pleura is involved in the disease.

d. *Derangements of the Circulation and Fever.*

If the hepatitis run an acute course, or be attended at an early period by inflammation of the serous membranes, it may be accompanied by febrile symptoms from its commencement to its termination. On the whole, however, this rarely happens; more frequently the fever attendant upon the inflammation disappears, to return at a later period in another form; or, as is especially apt to happen when the disease is latent, there is, at first, no excitement whatever of the vascular system, and it is only the formation of pus which gives rise to rigors, increased frequency of pulse and temperature of skin towards evening, night-sweats, &c.[1] In the last-mentioned cases, the fever presents the hectic form from the first, and this happens not unfrequently before there has been any local symptom to indicate the seat of the suppuration. According to the experience of Annesley, Haspel, Rouis and others, the fever frequently presents all the phenomena of a true intermittent, of a quotidian, tertian, quartan, or, occasionally a double type. As the disease advances, the attacks are wont to come on irregularly, sometimes several in the course of one day; and this circumstance, together with the inutility of Quinine, indicates their real nature.

Independently of these pseudo-intermittents, true intermittents are said by Haspel (*op. ant. cit.*, p. 274) to accompany hepatitis as complications,—a statement which is the more intelligible, when it is recollected that the miasmatic effluvia, which give rise to intermittent fever, dysentery, &c., exercise an important influence over the development of hepatitis.[2]

The fever accompanying hepatitis rarely presents severe nervous symptoms; delirium, somnolence, &c., only make their appearance a short time before death.

Even the nutrition does not by any means always suffer to a remarkable extent. It is true, that a state of exhaustion is gradually developed in most cases; but cases are met with where, notwithstanding the existence of abscesses in the liver, there is no emaciation, and, indeed, Rouis (*op. ant. cit.*, p. 119) records three observations, in which a great increase of the adipose cellular tissue was observed to take place, under such circumstances.

The above are the most important of the symptoms, which accompany the development and subsequent progress of suppurative hepatitis. We must not, however, expect always to meet with the entire train of symptoms perfectly developed; this is rarely the case; they are usually observed either in whole or in part, at certain stages of the disease only; or, indeed, all symptoms indicative of a local lesion may be entirely absent. According to the observations of Rouis (p. 107), collected in Algeria, the symptoms were perfect in only 8 per cent. of the patients who came under his notice, imperfect in 79 per cent., while in 13 per cent. the disease ran a latent course. These results show the difficulties, which,

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[1] Fouquier (*Gaz. des Hôpitaux* 16th Nov., 1841) observes, that it is a peculiarity of parenchymatous organs, and particularly of the liver, to occasion no fever, even when they are the seat of intense disease.

[2] Galen long ago expressed the opinion: "Tertianas semper fieri jecore laborante." Baillou and Senac misplaced the seat of intermittent fevers in the liver.

under certain circumstances, embarrass the diagnosis of suppurative hepatitis.

Among Rouis' cases, the symptomatology of the different forms of hepatitis, according to the more or less rapid progress of the inflammation, may be classified as follows:—

		Number in 143 Cases.	Number per cent.
Hepatitis acuta	Accompanied by all the peculiar symptoms	11	8
	By only a portion of these symptoms	15	10
Hepatitis subacuta	A well-defined commencement; obscure progress during the middle period; well-marked symptoms at the commencement of suppuration	6	4
Hepatitis chronica	Undefined symptoms, or none at all, prior to suppuration	62	44
	Undefined symptoms during the entire progress of the disease	30	21
	Progress masked, or latent	19	13

The symptoms ushering in the commencement of the disease were very various. Of the 143 cases, 80 commenced with symptoms of dysentery, 14 with those of gastric or gastro-enteric catarrh, 1 with those of gastralgia; 5 commenced under the form of irregular intermittent fever, and 5 were perfectly latent. In 21 cases, the symptoms of simple acute hepatitis, or of acute hepatitis complicated with a tendency to dysentery, were present from the first; while in 17, there were the symptoms of subacute or chronic hepatitis attended by dysentery.

It still remains for us to consider the symptoms, attendant upon the opening of the abscesses in different directions.

1. The abscess forces its way through the thoracic or abdominal wall. In this case a flattened tumor is slowly developed, which becomes fluctuating and red, and ultimately opens; the surrounding tissues are almost always œdematous. The communication with the purulent deposit in the liver is usually effected by the pus first escaping into the bands of areolar tissue between the liver and the abdominal wall, and then forcing its way outwards; the locality usually selected is the space below the ensiform cartilage of the sternum. In other cases, a swelling, which, like that of hernia, may be replaced, projects through the abdominal wall or an intercostal space, becomes fluctuating, points and ultimately bursts. Large abscesses sometimes press forwards a layer of the glandular substance, the thickened capsule of the liver and the aponeuroses and muscles of the abdominal walls, and in this way form extensive tumors, which push the ribs outwards and fill up the intercostal spaces, or produce a bulging in the hypochondrium or epigastrium. When the bulging is in the epigastrium, it is often the seat of pulsations propagated from the adjacent heart. More rarely it happens, that the pus burrows extensively within

the abdominal walls, before it appears externally; it may ascend beneath the serratus muscle to the axillary cavity, or the abscess may open close to the lumbar vertebræ, in the inguinal region, or in the inner side of the thigh. Rouis, moreover, states, that the pus from the liver may penetrate between the folds of the suspensory ligament, and afterwards be discharged at the umbilicus.

2. The abscess opens into the intestinal canal, or into the bile-ducts. In cases of this nature, the opening is not preceded by any well-defined symptoms; it is only when the abscess bursts into the stomach, that we meet with premonitory symptoms indicative of compression of this organ. The rupture into the stomach is followed by purulent vomiting or purulent stools, or sometimes by both.[1] When the abscess opens into the bile-ducts or the duodenum, it is often difficult to recognize the pus in the evacuations from the bowels, which is easily done when the contents of the abscess escape into the colon.[2] In many cases, the patients are not cognizant of the occurrence of the rupture, while in others, attention is drawn to the circumstance by a sudden abatement of the pain and dyspnœa, &c.

3. The abscess opens into the pelvis of the right kidney, and the pus escapes with the urine. This is a very rare event mentioned by Annesley, and is preceded by no well-marked symptoms.

4. The abscess empties its contents into the bronchial tubes. This event is usually preceded by the signs and symptoms of inflammation of the base of the right lung, which rarely extends higher up than a few inches: these signs are dulness, bronchial breathing, rusty expectoration, pains, &c. The pains cease, but the dulness remains, and the respiratory murmur at some places becomes quite inaudible, until suddenly a large quantity of thick, whitish, or reddish-brown, pus is expectorated, sometimes mixed with sanguinolent fluid or with shreds of the hepatic pulmonary tissue. After a time, even pure bile, which has escaped from the open bile-ducts into the cavity of the abscess, is occasionally expectorated. In one case, Rouis collected 900 grammes (29 oz. troy) of bile in twenty-four hours. In most cases of this nature, the breath acquires a fœtid odor. When the cavity in the lungs is superficial and empty, the phenomena of cavernous breathing may be observed. After a time, the quantity of expectoration diminishes, it becomes mucous, and by degrees ceases entirely, while the process of reparation gradually advances. But the curative process is often unsuccessful; the difficulty of expectorating the discharge increases more and more; the larger the cavity is, the more its walls are kept asunder by adhesions, and the more extensive is the destruction of the diaphragm. Here death ensues, under symptoms of hectic fever.

5. The abscess opens into the right pleural cavity. This event usually takes place without occasioning any remarkable disturbance; a dull pain and slight dyspnœa, together with the physical signs of pleuritic effusion are the sole indications of its occurrence. After the pus has entered the pleural cavity, it may still escape externally through the wall of the chest or through the bronchi. In the former case, a flattened tumor is developed in one of the intercostal spaces, which opens externally in the

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[1] Morehead observed one case, where there was no vomiting, and where no pus could be detected in the stools, after the abscess had burst into the stomach.

[2] See an observation by Dr. Murchison,—*Transactions of the Pathological Society of London*, Vol. VIII.

same way as in ordinary empyema; in the second case, the substance of the lung is eroded, and the pus passes into the bronchial tubes with the symptoms above described.

6. The abscess has been observed, by Rokitansky, Graves, and Fowler, to burst into the pericardium, an event which is announced by violent pains, a feeling of suffocation, and the physical signs of very rapid pericardial effusion. After a short period, the case terminates fatally.

7. When the pus from an hepatic abscess is poured into the peritoneal cavity, the symptoms of acute peritonitis immediately ensue, which usually proves fatal in a few hours or days. In some cases, the symptoms are less violent, namely, when the quantity of pus which escapes is at first small, and its extension over the entire peritoneum is prevented by means of adhesions. In this case, circumscribed collections of pus are formed in the peritoneal cavity, which may open externally, either directly through the abdominal wall at the epigastrium (Rouis, *op. cit.*, p. 144), or between the eleventh and twelfth ribs (Cambay, *op. cit.*, p. 225), or through the inguinal canal (Haspel, *op. cit.*, p. 193). In all such cases hitherto observed, with the exception of two mentioned by Graves, death by exhaustion ensued. The evacuation of these collections of pus into the intestine, the bladder, &c., such as happens not unfrequently in the case of other circumscribed peritoneal exudations, has not as yet been observed to take place in the cases under consideration.

Independently of the peritonitis resulting from perforation of the hepatic abscess, suppurative hepatitis may give rise to inflammation of the peritoneum, by the propagation of the inflammatory process from the liver to the adjoining serous membrane; moreover, this peritonitis may be general, and induce death by the abundant exudation. In like manner, pleurisy and pericarditis may be developed by a propagation of the inflammation, without any perforation. (Morehead, *loc. cit.*, p. 352 *et seq.*)

As regards the frequency with which hepatic abscesses open, and the relative frequency of the different directions in which the opening takes place, we possess statistical data furnished by several observers, which although they do not entirely correspond, enable us, at all events, to form a conclusion approximating the truth. According to the observations of Rouis, which have been carefully analyzed with this object in view, among 162 cases terminating fatally, there were 96 in which the suppuration had not passed beyond the boundaries of the liver; 16, where there were several abscesses present, of which a portion only had burst, and lastly, 50, where the contents of the abscess had escaped beyond the liver. Of the abscesses which had passed beyond the boundaries of the liver, there were in the first place 6, the purulent contents of which had merely come in contact with the neighboring organs, without destroying them, viz.: with the right half of the already attenuated diaphragm, with the pericardium, with the posterior surface of the stomach, with the pancreas, with the gall-bladder, and with the colon, the coats of which were destroyed as far as its mucous membrane; 26 abscesses had evacuated themselves into the adjoining closed cavities, viz.: 14 into the peritoneum, 11 into the right pleural cavity, and 1 into the pericardium. Among these there was a case where one abscess burst into the pleural cavity and another in the same liver opened into the peritoneum, and also a second case, where the pus was first circumscribed at the root of the great omentum and afterwards escaped externally at the epigastrium. In 30 cases, the abscess had forced its way externally, viz.:—

Through the abdominal walls in the hepatic region, in.....	2 cases.
Directly through the lung into the bronchi.....	15 "
After previously bursting into the pleural cavity.....	2 "
Through the stomach.....	5 "
Through the duodenum.....	1 "
Through the transverse colon.....	3 "
Through the bile-ducts.....	1 "
Through the gall-bladder.....	1 "
Total.....	30 "

In one of these cases, there were two abscesses in the same liver, one of which opened into the colon, and the other into the stomach.

In 17[1] cases, the abscesses were opened artificially.

Of 39 cases, in which complete recovery took place, there were:—

17, where the abscess had burst through the abdominal or thoracic wall, viz.: 3 in the last intercostal space of the right side, 13 in the epigastric region below the sternum, and one at the umbilicus.

15, where the abscess opened into the bronchi.

3, where it opened into the stomach, and

4, where the abscess poured its contents into the transverse colon.

Of 25 cases described by Haspel, an opening took place externally

through the thoracic or abdominal wall, in.....	7
into the lung, in.....	2
into the pleural cavity, in.....	4
into the cavity of the peritoneum, in.....	2
(in one of these cases, the pus passed down into the scrotum).	
into the stomach and small intestine.....	0
The abscess did not extend beyond liver, in.....	10

Of 10 cases recorded by Cambay, 1 opened into the cavity of the peritoneum, 1 into the gall-bladder, 2 into the bronchi, while 6 remained enclosed in the liver, and in one of these, there was commencing cicatrization.

Out of 140 cases, Morehead observed an opening to take place into the lungs or pleural cavity, 14 times, or in ten per cent.; into the stomach and intestines, 5 times, or in 3·5 per cent., and of these 3 recovered; in 2 cases only did the abscess open into the peritoneal cavity, and even in these cases there was no certain proof that the opening had taken place; on the other hand, secondary circumscribed peritonitis occurred 7 times, and secondary general peritonitis, 14 times, independently of any rupture of the abscess. In 5 cases it was necessary to assume, that the pus had been absorbed.

Of Andral's 11 cases, 9 abscesses did not extend beyond the boundaries of the liver, one poured its contents into the stomach, and another into the peritoneal cavity.[2]

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[1] The 17 here ought possibly to be 7; otherwise the number of fatal cases accounted for is 172 in place of 162.—TRANSL.

[2] The author does not appear to have met with the work of Mr. Waring, of the Madras Medical Service (*An Enquiry into the Statistics and Pathology of some points*

4. *Modes of Termination.*

Suppurative hepatitis belongs to the class of severe maladies, which imperil life, and which terminate in death far more frequently than in recovery. There is not a sufficient number of observations, to determine, with any degree of accuracy, how frequently in our own climate, the process terminates by one or the other mode; the data which have been collected in warm climates, and which have been subjected to statistical analysis, are far more comprehensive. The results of observations collected in different countries are, however, far from agreeing,—a circumstance which is at once explained, when it is recollected, that in addition to the hepatic disease, there are many other agencies of a local or general nature, and especially the dysentery which so often accompanies the hepatitis, that influence the mode of termination. I have collected here the results of the most important observations upon the point, in order to give some

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connected with Abscess in the Liver, as met with in the East Indies. Trevandrum, 1854. 8vo., pp. 206), in which 300 cases of hepatic abscess, which terminated fatally in India, have been collected from various sources. The reader will find much valuable information in Mr. Waring's memoir, respecting many of the points bearing upon abscess of the liver, which have been so ably discussed by Professor Frerichs. The following, for example, is an analysis of the modes of termination of the 300 abscesses :—

	No.	Per cent.
Remained intact, the abscess not extending beyond the boundaries of the liver	169	56·335
Evacuated by operation, a solitary abscess being present	29	
" " " there being numerous abscesses, one opened, and the others remaining intact	18	16·000
One abscess opened by operation, another subsequently bursting into the abdominal cavity	1	
Opened spontaneously into the thoracic cavity	14	4·666
" " into the right lung	28	9·333
" " into the abdominal cavity	15	5·000
" " into the colon or large intestines	7	2·333
" " into the stomach	1	
" " into the hepatic vein leading to the vena cava	2	
" " into the hepatic vein at its junction with the vena cava, and another abscess communicating with the cellular tissue around the right kidney	1	
Communicated with the hepatic ducts	1	
" with the right kidney	2	
" with the gall-bladder	1	
" with an abscess in the iliac region		6.333
Opened spontaneously through the ribs in the back	11	
One abscess had opened into the colon, and another had passed off by the hepatic ducts into the duodenum	1	
One abscess had opened into the stomach, a second into the duodenum, and a third had been evacuated by operation	1	
One abscess had opened into the abdominal cavity, and a second into the lungs	1	
Terminated in erysipelas of the lower extremities, simulating phlegmasia dolens, the abscess opening into the lungs	1	
Doubtful	5	
	300	100·000

—TRANSL.

idea of the manner in which the hepatitis of the torrid zone is wont to terminate.

Of 203 cases collected by Rouis (*op cit.*, p. 147), 162 terminated fatally; in 39 there was a complete, and in 2, an imperfect cure; thus the cures were 20 per cent., and the deaths 80 per cent. The fatal termination was brought about in various ways:—[1]

By the severity of the local disease, or by the concomitant dysentery	125	times.
By gangrene of the walls of the abscess	3	"
By peritonitis propagated from the hepatic inflammation	3	"
By the bursting of the abscess into the peritoneal cavity	12	"
By rupture of the adhesions between the liver and the abdominal wall	2	"
By the escape of pus into the pleural cavity	11	"
By the passage of pus into the pericardium	1	"
By intercurrent pneumonia	2	"
By the extension of the pneumonia induced by the passage of the pus into the bronchi	3	"
Total	162	"

As regards the influence of dysentery upon the progress of the local affection, and upon the mode of termination of the disease, experience shows that the abscesses which are complicated with dysentery, open externally and cicatrize, less frequently than the uncomplicated cases. Of 24 abscesses uncomplicated with dysentery, 19 discharged their contents externally, and of these, 14 recovered, viz.: 4 (out of 5 cases) where the opening was in the wall of the abdomen or chest, 6 (out of 8) which burst into the bronchi, and 4 (out of 6) which opened into the digestive organs. Of 118 abscesses complicated with dysentery, only 59 opened externally, and of these there recovered 13 out of 29 cases, where the opening was in the abdominal walls, 9 out of 22 cases, which burst into the bronchi, and 3 out of 8 cases, which discharged their contents into the intestinal canal. Thus, of the uncomplicated cases, there were 14 recoveries among 19 cases, which opened externally, whereas of the cases complicated with dysentery there were only 25 recoveries in 59 cases; in other words, of the simple abscesses 80 per cent. opened, and 60 per cent. recovered, but of the complicated cases only 50 per cent. opened, and 29 per cent. recovered.

The observations, collected by Morehead in the East Indies, do not agree with those of the French physicians in Algeria. On the whole, Morehead found the rate of mortality much smaller.[2]

[1] Rouis makes no mention of the fatal termination by pyæmia, which, according to my experience, is occasionally induced by consecutive inflammation of the hepatic veins.

[2] The mortality in the European General Hospital at Bombay, amounted to 14·1 per cent.; or, 711 cases admitted between the years 1838 and 1853, with acute and chronic hepatitis, 102 died. In the Jamsetjee Jejeebhoy Hospital the mortality was 34 per cent.; of 208 cases admitted for acute diseases of the liver, 23 died, or 11 per cent., while of 198 cases admitted for chronic hepatic affections, 102 died, or 51·5 per cent. Morehead, however, observes, that cases of cirrhosis must have been included under the terms acute and chronic hepatitis.

We possess no statistical data of any value for forming a conclusion, as to the results of hepatic inflammation in our own climate. So far as we are able to judge, endemic hepatitis appears to be less dangerous than the tropical form. This remark applies especially to the traumatic cases; the cases where the hepatitis is induced by the penetration of round worms into the bile-ducts, or by pyæmia, as a rule, terminate fatally, as do likewise the cases which arise from an obstruction to the flow of bile, or from biliary concretions.

In the cases which terminate in recovery, convalescence is always tedious. Months or even years may elapse, before the bodily nutrition is restored, and the patients recover their strength; and even then the restoration is very often incomplete. The digestion continues for a long time deranged, partly on account of defective secretion of bile, and partly because the movements of the stomach and intestine are impeded by numerous adhesions. In rare cases, even the cicatrization of the cavity of the abscess is imperfect. Morel recorded one case, where a sero-purulent fluid continued to flow from the wound in the right side, for three years after the opening of the abscess. Casimir Broussais has given the details of another case, where the cicatrix in the epigastrium had to be punctured almost every two months. The operation was repeated twenty-four times, in less than four years. On each occasion, a glassful of thick pus escaped and the opening closed up again. The cause of this anomaly must be attributed to sinuses and cavities, with dense, callous, not easily approximated walls.

The question has been raised whether abscesses of the liver, which have not discharged their contents, can undergo a cure by absorption of the pus. Rouis doubts the possibility of such an event, whilst Haspel (*op. cit.*, p. 240), Catteloup, Cambay (*Dyssenterie de la Province d' Oran*, p. 223, Obs. 34 and 37), and particularly Morehead (*loc. cit.*, p. 346) record observations, which, in their opinion, demonstrate the possibility of this mode of cure. The question is one to which it is difficult to give a positive answer. Analogy is certainly in favor of the possibility of such an occurrence, and there are many anatomical appearances which scarcely admit of any other explanation. It is true, that we must beware of regarding every radiated cicatrix enclosing cheesy matter, met with in the liver, as a cured abscess; we shall subsequently see how often appearances of this sort accompany constitutional syphilis. Still, there are observations such as those of Catteloup, Cambay, C. Broussais and Morehead, where the appearances can only be regarded as the remains of true suppurative hepatitis. In these cases, there were not merely all the symptoms of suppurative hepatitis going before, but the cicatrices and the size of the entire organ, indicated a loss of substance, such as occurs in abscess, but not in syphilitic affections of the liver.

Another question is, whether all the cases, where there are several abscesses in the liver, terminate fatally. Speaking generally, the question may be answered in the affirmative, although exceptions undoubtedly occur. Small abscesses are particularly apt to become encysted for a long period, without giving rise to any great inconvenience. Thus Budd (*loc. cit.*, p. 111) states, that his colleague, Mr. Lawson, followed his profession as a Surgeon, for ten years after an attack of hepatitis, which left behind several abscesses. Casimir Broussais likewise records an observation, where four cicatrices were found in the liver of a man, who had survived an attack of dysentery and hepatitis. In this case, however, there is still the doubt, whether the cicatrices are to be regarded as the remains of

abscesses, or whether the connective tissue was the direct result of the inflammation.

5. *Duration of the Disease.*

The question, whether hepatitis is to terminate in death or recovery, is rarely decided in a few weeks; months usually elapse before either the one or the other result takes place.

Rouis, who has carefully analysed his extensive materials, in order to obtain some information upon this point, has arrived at the following results:—

The cases which terminate fatally, lasted on an average:—

A.	When the abscesses did not open externally.....	70 days
B.	When the abscesses discharged their contents externally:—	
1.	Through the abdominal or thoracic wall.....	70 "
2.	Directly through the bronchi.....	125 "
3.	Through the bronchi after previous discharge into the pleura.....	185 "
4.	Through the stomach.....	150 "
5.	Through the colon and bile-ducts.....	some months.

The average duration of the cases included under series B, was 110 days.

The duration of the cases which recovered was as follows:—

1.	Where the abscess burst throught the thoracic, or abdominal wall.....	140 days.
2.	Where the abscess burst through the bronchi.....	115 "
3.	" " " through the colon.....	140 "
4.	" " " through the stomach....	180 "

The average duration was 140 days.

There are cases, the duration of which exceeds the limits just mentioned. Thus Andral records a case of hepatitis produced by a blow on the right hypochondrium, which did not prove fatal until after two years. On the other hand, cases of hepatitis have been observed, both in our own climate and also in the tropics, running a much more rapid course, whether they proved fatal or not. When the progress is very rapid, the pus is sometimes found infiltrated through the grayish-yellow, softened parenchyma of the liver—death, in fact, having taken place before an abscess had been formed. Haspel (*loc. cit.*, p. 355) records a case of this nature, under the title of "Ramollissement avec infiltration de pus;" and Rouis has also repeatedly observed pus infiltrated through the hepatic tissue.

6. *Complications.*

With the exception of dysentery and the diseases consequent upon the hepatitis, complications are not of frequent occurrence. The most common is an attack of intermittent fever accompanying the commencement of the inflammation, which, at the supervention of suppuration, but not before, gradually loses its typical character. To this are due the

dense splenic tumors, which are not uncommonly observed in tropical hepatitis. Suppurative hepatitis is likewise occasionally found complicated with induration and cirrhosis of the liver,[1] tubercle of the lungs, chronic ulcer of the stomach (Rouis), chronic nephritis (Cambay), &c. These complications are, so far, of importance, inasmuch as they accelerate the exhaustion of the patients.

7. *Prognosis.*

The prognosis is in general unfavorable. Even when the disease apparently takes a hopeful course, it must not be forgotten that dangerous symptoms may suddenly make their appearance, because we are completely ignorant of the number of the abscesses, of the direction in which they may open, &c. Speaking generally, the chances of recovery are impaired:—

1. by the existence of dysentery as a complication;
2. by obstinate intermittent fever, inducing a state of cachexia;
3. by symptoms of peritonitis.
4. Cases where the abscess bursts into the pericardium or the peritoneum are always fatal. Hence the saying of Hippocrates is true: "Lethales sunt illi abscessus qui effundunt in intro."
5. Most cases, where the abscess bursts into the pleural cavity, terminate fatally; and the same remark applies to
6. Extensive hepatization, or purulent infiltration of the lungs.

The progress is more favorable when the abscess discharges itself through the abdominal wall, or into the colon, the bronchi, or bile-ducts.

8. *Diagnosis.*

There are forms of hepatitis which run such a latent course, that it is impossible to recognize them; others, again, are accompanied by such marked symptoms that they cannot be overlooked. In most cases, a correct diagnosis will only be arrived at, by not relying upon individual symptoms, by taking a general view of the mode of origin and entire clinical history of the case, and after excluding, by comparison, the diseases of the liver and of the neighboring parts, which may give rise to symptoms similar to those of hepatitis.

Of diseases of the liver, there are, in the first place, the serous cysts and echinococci, which are apt to be mistaken for abscesses. They are easily distinguished by their slow growth, by the absence of pain and febrile symptoms, and, likewise, by the absence of impaired nutrition. Echinococci, passing into suppuration, may give rise to symptoms bearing a close resemblance to those of hepatic abscess. Here an accurate diagnosis can only be arrived at by carefully considering the antecedent history. Cancer of the liver can only cause a mistake in those cases where a large, white medullary mass imparts to the fingers a feeling of

[1] The circumference of the abscesses is often extensively indurated; and cases are also met with where they are developed in cirrhotic livers. Morehead observed this five times. Budd (*op. cit.*, p. 106) formerly doubted the possibility of such a coincidence.

fluctuation. It is distinguished, however, by its different clinical history, by the lengthened duration of the tumors without any fever, and by the existence of other smaller and hardened nodules, appreciable to palpation. Diseases of the gall-bladder, and especially inflammation and distention of the bladder with fluid, may far more readily lead to mistakes. In distinguishing these affections from hepatic abscess, we must trust to the situation, the pear-shaped form, and the mobility of the tumor, as well as to its constantly soft, fluctuating character, and to the circumstances of its not having been preceded by any induration. Moreover, the distended gall-bladder scarcely ever contracts adhesions to the abdominal walls;[1] the abdominal walls themselves are not œdematous; the liver is not enlarged; there is no hectic fever; and the disease of the gall-bladder is frequently preceded by the colic of biliary calculi.

Peri-hepatitis in many cases bears a resemblance to hepatitis vera, but there is no enlargement of the liver; there is never any circumscribed hardness; the general derangements are insignificant and the progress is much more rapid.

Under certain circumstances, it may be difficult to distinguish the pneumonia and pleurisy of the right side, resulting from the extension of a hepatic abscess into the right side of the chest, from simple pneumonia and pleurisy, especially when the hepatitis is latent at its commencement. In such a case, we can only avoid falling into error by carefully studying the previous history. After the passage of the pus into the bronchi, the diagnosis is in most cases easy, because the quality of the expectoration, and the occurrence in it of disintegrated hepatic tissue or of bile, will indicate its origin. The difficulties encountered in the time of Baglivi and Stoll, in distinguishing hepatitis complicated with cough, from pleurisy and pneumonia, can no longer be experienced by any one who has had any experience in physical diagnosis.

The earlier physicians enumerated a number of symptoms, by means of which they believed they could ascertain the site of the inflammation, whether it was in the convex or the concave portion, the right or the left lobe, of the liver. These symptoms, which referred mainly to the nature of the pain, the accompanying derangements of the respiration or digestion, the character of the pulse, &c., are uncertain in their indications. The question can only be answered by changes in the form of the liver of such a nature as to be appreciable by percussion and palpation, and occasionally also, by the sensation of resistance imparted by the seat of pain.

9. *Treatment.*

A series of general remedial measures have been employed in the treatment of hepatitis vera, which we must first examine, before proceeding to sketch a plan of treatment adapted to the individual forms of the disease.

a. *General and Local Abstractions of Blood.*

In ancient, as well as in modern times, phlebotomy has repeatedly been recommended, as the most certain measure for limiting the pro-

[1] I have observed this in only one case, which I treated at Breslau along with Dr. Klose. Here the bladder was punctured, with a favorable result.

gress of hepatitis. It is long, however, since Van Swieten brought forward objections to this plan of treatment, which the experience gained in hot climates has not tended to dispel. Venesection is still less adapted to cut short the morbid process in hepatitis, than it is in pneumonia; and, at a later stage, when suppuration supervenes, the state of cachexia, usually imperilling the life of the patient, ought to render us cautious of its inconsiderate employment. Here, as in inflammation of the lungs, it is difficult to determine what influence venesection can exert upon the local disease; no great value can be assigned to the practise as a means of alleviating the subjective symptoms, and that it does not prevent suppuration, is a fact. It follows, that blood-letting must only be ventured upon in cases of traumatic hepatitis, and in robust plethoric individuals, where there is great tenderness together with remarkable enlargement of the liver, urgent dyspnœa, &c. Here, venesection, by alleviating the respiratory symptoms, and freeing the circulation, may react favorably upon the local affection. Under other circumstances, and particularly in all cachectic diseases, such as dysentery, &c., blood-letting must be abstained from.

Local abstractions of blood are practised with greatest advantage upon the fundament; at all events, they act here more directly upon the portal circulation than in the hepatic region; the latter locality, however, is to be preferred, when our object is to overcome an attack of peri-hepatitis or local peritonitis. In such cases, advantage will be derived from the simultaneous application of warm cataplasms, and afterwards, from the inunction of mercurial ointment. Tepid baths will also be found serviceable, in cases where the pain and the fever permit their employment.

b. *Purgatives.*

Purgatives are particularly applicable to cases where the intestinal functions are sluggish, whilst dysentery, as a rule, forbids their employment. The increased secretion of bile to which they give rise, as well as the derivative effect of the augmented intestinal secretion, may be expected to exercise a favorable influence upon the circulation in the liver. First among medicines of this class, is Calomel, which has been spoken of in equally commendatory terms, by Lind, Annesley, Haspel, Morehead, Rouis, and others. In employing this remedy, care must be taken to avoid salivation. With this object, Haspel, Rouis, and others, administer a scruple in one dose daily, or they order a dose of Infusion of Senna or Castor Oil to be taken before the Calomel. When there is no constipation, small doses are preferable; and when the hepatitis is complicated with dysentery, the French physicians adopt Segond's plan of combining the Calomel with Ipecacuanha and Opium. In cases where there is severe fever, Rouis recommends a combination of Calomel and Digitalis.

Calomel is contraindicated where there is great irritability of the stomach, and also after the supervention of suppuration, and in all cachectic conditions of the system.[1]

In addition to mercurial preparations, we may have recourse to the salines, Castor Oil, and the allied mild purgatives, which are often indis-

[1] Budd objects to the employment of mercury, on the ground that the abscesses are developed so rapidly, that the remedy has not time to take effect, and that its employment is always injurious when suppuration has already taken place.

pensable, because by persisting in the use of Calomel, there is danger of inducing salivation. In the more advanced stages of the disease, it is better to avoid all remedies calculated to produce exhaustion, and to employ Rhubarb, Senna, Aloes, and similar remedies in place of the saline purgatives.

c. *Emetics.*

Emetics undoubtedly exert a powerful influence over the circulation of blood in the liver and upon the secretion of bile, because during the act of vomiting the organ is compressed on all sides by the abdominal walls. But, on that very account their employment should be restricted to the early stages of the disease; in cases where inflammatory deposits or abscesses already exist, they may be productive of great injury. They are likewise to be avoided, when the mucous membrane of the stomach is in an irritable condition. They are particularly serviceable when the hepatitis is accompanied by gastro-enteric catarrh, and in cases characterized by painless, hyperæmic enlargements of the liver, remaining obstinately stationary for a long period. Antimony or Ipecacuanha is to be selected, according to the condition of the intestinal secretions.

d. *Revulsives.*

Epispastics applied over the region of the liver, in the form of large, or, of (what in my experience are preferable) small, oft-repeated blisters, are suitable in cases, where, notwithstanding the protracted use of antiphlogistics, the local affection becomes chronic, and threatens to pass into suppuration. Haspel (*op. cit.*, p. 297) has observed dense inflammatory deposits, diminishing and gradually disappearing under their use.

e. Opium, Quinine, Cinchona Bark, Steel, &c., are remedies, which are usually indispensable in the treatment of hepatitis. They exert no influence, however, over the local affection, and they are merely serviceable for counteracting symptoms.

In the treatment of individual cases of hepatitis, due regard must be had to the more or less acute character of the inflammation, to its causes, to the constitution of the patient, and lastly, to the existence of complications, more particularly of dysentery.

In the acute forms of the disease in robust individuals, local blood-lettings can seldom be dispensed with. From ten to fifteen leeches should be applied to the anus, or, if necessary, to the seat of pain; and when the hyperæmic swelling and the pain in the liver are considerable, and the dyspnœa urgent, venesection must be had recourse to. The hepatic region ought to be covered with warm cataplasms. At the same time, it is well to prescribe internally a few large doses of Calomel, the action of which is to be kept up by means of salines or Castor Oil. When the fever is very severe, Digitalis may be combined with the Calomel, as recommended by Rouis.

When the hepatitis is accompanied by intense gastric catarrh, thickly-coated tongue, nausea, &c., the best results may be expected from an emetic, provided that the inflammation is not too acute, or that it has been previously subdued by the abstraction of blood. Emetics are also suitable in those cases, where the swelling remains stationary after the employment of antiphlogistics.

In such cases as those last mentioned, blisters are also serviceable.

Not unfrequently, the hepatitis is accompanied by nausea, vomiting, distention and tenderness of the epigastrium, together with other symptoms of hyperæmia of the mucous membrane of the stomach, and then both Calomel and emetics are contraindicated. In cases of this nature, the rule is to subdue the irritability of the stomach by local blood-lettings and narcotics, before having recourse to the milder laxatives.

When dysentery has preceded the hepatitis as a complication, we must refrain from general blood-letting, purgatives and emetics, and restrict ourselves to the application of leeches to the anus, or of cupping-glasses to the abdominal parietes along the course of the colon, the abdomen is to be covered with warm cataplasms, and a solution of Gum, or Segond's pills, composed of Ipecacuanha, Calomel and Opium, are to be administered internally.

When the dysentery is persistent, we must endeavor to check the discharge from the bowels by means of opiates, Rhatany, Tannin, Alum and similar astringents.

In the subacute forms of the disease, we must avoid general blood-letting and strong purgatives. Here, it is best to restrict ourselves to the employment of an emetic, followed by a gentle laxative. When the case is complicated with chronic dysentery, Rouis particularly recommends Calomel in doses of two or three grains several times repeated, and followed by Opium.

In the chronic forms, care must be taken to exclude all remedies having a weakening tendency. If the intestinal functions are deranged, the bowels may be opened by mild salines and afterwards by Rhubarb, and along with these we may employ warm cataplasms and tepid baths, and at a later period, epispastics. When, however, there is obstinate constipation, Calomel must be given before the salines. In the cases complicated by dysentery, the French physicians recommend small doses of Calomel, alternating with Opium, and afterwards Opium along with astringents. Under such circumstances, a mild nutritious diet is indispensable for keeping up the patient's strength. When this treatment proves unsuccessful, the only chance of safety is to be looked for in a change of climate; and with this it is well to combine, when possible, the use of the warm alkaline mineral waters, such as those of Vichy,[1] Ems and similar springs.

The Treatment of Hepatic Abscesses and of the diseases to which they give rise.

If the therapeutic treatment adopted to combat the inflammation does not succeed in preventing suppuration, care must be taken not to lose time in counteracting the exhaustion, which at this period is apt to supervene in an imminent degree. Antiphlogistics must not be persisted in too long; and, in cases where they fail to take effect, we must restrict ourselves to the use of Morphia and the preparations of Hydrocyanic Acid, and substitute, at an early period, a tonic course of treatment.

When the pus makes its way to the lung and the symptoms of pneumonia supervene, it is best to confine ourselves to the use of Digitalis. After the cessation of the fever, blisters may be employed, but are seldom

[1] For the mineral constituents of these springs, see Vol. I., p. 88, *note*.—TRANSL.

of much service; narcotics, and particularly Morphia and Opium, are the only remedies which give relief.

When the abscess opens into the abdominal cavity, death is almost inevitable; in such cases, opiates, warm cataplasms and absolute rest in the decumbent posture, are indicated, solely with the object of alleviating the symptoms and of favoring the limitation of the exudation.

A similar practice is to be recommended when the pus finds its way into the cavity of the pleura.

When the abscess takes a direction outwards, we ought not to delay in making an artificial opening. In most cases, when fluctuation can be detected, the abscess has already attained a considerable size; and the longer its evacuation is delayed, the greater are the dangers of its bursting into the abdominal cavity, of an extensive destruction of the liver, and of the formation of a dense, rigid, not easily cicatrized cyst. Indeed, we must not always wait for the supervention of fluctuation, or for the œdematous infiltration of the abdominal walls; because these signs, especially in the intercostal spaces, are sometimes late in making their appearance; in such cases, the prominences of the false ribs and the obliteration of the intercostal spaces suffice to justify the operation.[1] In having recourse to it, care must be taken to prevent the entrance of pus into the abdominal cavity,—an indication which is best fulfilled by following the methods recommended by Bégin and Récamier. Bégin places the patient on his back, with the upper part of the body bent forwards, and the thighs flexed upon the abdomen, and after having accurately determined the limits of the abscess, which the thinning of the abdominal walls and the fluctuation enable us to do, he makes an incision from 6 to 8 centimètres (2⅓ to 3¼ inches) in length, dividing the skin, the subcutaneous adipose tissue, the muscles and the aponeuroses. The peritoneum is then opened, as in the operation for hernia, and slit up upon a grooved director, to the same extent as the primary incison. The wound is then dressed with charpie. After three days, when the dressing is removed, the liver is found to have contracted firm adhesions to the margins of the wound, so that the abscess may be opened without apprehension. The operation is easily performed, and perfectly certain; only, as Haspel and Rouis have justly observed, we can scarcely expect that a firm adhesion should be formed, when the abscess does not produce any elevation of the abdominal walls, because it is possible that the tumor may not project into the opening of the wound.

Récamier recommends the application to the most prominent part of the swelling, of four or six grains of Caustic Potash, so as to produce a

[1] Budd is of an opposite opinion. He advises that the abscesses be not opened, but be left to nature, for fear of the entrance of air giving rise to dangerous suppuration. But the entrance of air is not prevented by a spontaneous opening, which, moreover, incurs the dangers of delay indicated above. Morehead advises (*op. cit.*, Vol. II., p. 110), that in the case of small abscesses pointing in the epigastrium, we should wait until the skin becomes red, and then make an opening with a bistoury. When there is general prominence of the right side of the thorax, he thinks it matters little whether an opening be made or not, after fluctuation can be felt; gangrene of the soft parts and caries of the ribs can then seldom be avoided. If the liver project several inches beyond the margin of the ribs and present obscure fluctuation, or if the abscess be large, with extensive fluctuation, there is danger, according to him, of inducing gangrene and irritative fever, by making a puncture with a large trocar, or with a knife, and it is better to employ an exploratory trocar. In such cases, he recommends the puncture to be made carefully, so as to exclude the air, and that it be repeated frequently, so as to evacuate the pus gradually.

slough of 3 or 4 centimètres in diameter (about 1 to 1½ inch). After the separation of the slough, he applies a stick of caustic potash to the resulting sore, and repeats the application from three to six times, until the abscess opens. The apprehensions entertained by Boyer, Velpeau and Cruveilhier, that the application of the caustic might give rise to peritonitis, have not been confirmed. The operation is certain; but a long time elapses before the object is attained, and a loss of substance in the abdominal walls is the result.[1]

A simple puncture is only permissible, when the pus has already penetrated through the superficial layer of the abdominal aponeurosis, or the intercostal muscles; in other cases it ought to be avoided as dangerous.[2]

After the abscess has opened, the evacuation of the pus ought to be left to muscular contraction, and strict rest must be enjoined, because any imprudent movement might rupture the adhesions, and give rise, as Rouis has observed, to sudden death. The dressings should consist of large poultices; and, when the discharge becomes serous, a pledget of charpie may be applied. When the cavity is of large size, the base of the thorax may be compressed by means of bandages, and it may also be of service to syringe it out with tepid water.

The indications for internal treatment consist in maintaining the patient's nutrition and keeping up the strength, by means of Quinine, Steel and a suitable diet.

Sometimes the cavity is a long time in closing up, and continues to discharge a sero-purulent fluid, and then, according to the experience of Rouis, the cure is prevented by an inflammatory enlargement of the liver. To counteract this, Rouis recommends the internal, as well as the external use of the sulphureous mineral waters, which he believes to be much more certain in their results than purgatives, blisters, or Iodine injections.

In cases where the abscess opens externally of its own accord, the indications for treatment are more simple. When the opening takes place through the abdominal parietes, the skin frequently becomes undermined by extensive sinuses, requiring incision.

When the abscess opens into the intestinal canal, the bile-ducts, or the right kidney, strict rest is to be enjoined, so as to favor the chances of adhesions.

When the pus escapes by the bronchi, we must prescribe opiates, which experience shows to afford the greatest relief. Besides this, care must be taken to maintain throughout the strength of the patient. The remains of pleuritic effusions, and of inflammatory exudations into the lungs may be left to nature, or, if they become stationary, they may be treated with blisters and diuretics.

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[1] There is nothing in the operative methods, recommended by Graves, Horner, Vidal and Cambay, to entitle them to any pre-eminence over those of Bégin and Récamier.

[2] Mr. Waring (see p. 147, note) has collected the particulars of 81 cases in which hepatic abscess was opened in India. The deaths were 66 and the recoveries 15. In all of them, the abscess would appear to have been opened by a simple puncture with a trocar or bistoury. In 58 of the 81 cases, the mode of operating was specified. Of these, the trocar only was used in 23 cases, the exploratory needle, followed by the trocar in 19, and the lancet or scalpel in 16 cases. No mention is made of the adoption of any measures to promote adhesions, previous to opening the abscess.—TRANSL.

Hepatitis Syphilitica.

Syphilitic Disease of the Liver.

1. *Historical Account.*

That the liver becomes involved in the course of syphilis, is an opinion which is as old as the history of syphilis itself. Biased by the doctrines of Galen, medical men formerly believed that syphilitic ulcers were the result of a corruption of the humors, the origin of which was to be looked for in the liver, which had become diseased from the action of a volatile contagious principle. This explanation was already announced by Hutten in his work, *De Guaiaco* (Cap. III.), and was given in detail by Fallopius in his *Tractatus de Morbo Gallico* (Cap. X. to XIII). The latter, after refuting other theories, arrives at the following conclusion; "Si igitur hoc perpetuum est—actiones læsæ et sanguis a statu naturali recedens—ideo necessarium est, hunc morbum afficere fontem foventem hanc facultatem, et hoc est hepar, in quo, tanquam in propria parte, oritur morbus." He likewise appeals to similar views held by Ant. Musa, Brassavolus, Montanus, Ant. Gallus and others.

Almost simultaneously with this idea of a primary syphilitic disease of the liver, there arose another, according to which the liver first became affected consecutively to the disease of the genital organs, in consequence of the gradual development of a corrupt condition of the humors. This view was maintained by Cutaneus (*in Aloysius Luisinus de Morbo Gallico.* Venet., p. 151), and also by Vella (*ibid.*, p. 207) and by Alph. Ferro (*ibid.*, p. 433): "Quibus infectis (pudibunda scilicet) vitiantur venæ capillares, deinceps magnæ venæ atque arteriæ nec non et hepar ipsum et reliqua principalia membra." The idea of a primary syphilitic affection of the liver was likewise combated by Botalli, Petronius (*De Morbo Gallico*, Lib. I., Cap. XVII.) and Borgarucius, the last of whom had found the liver healthy on *post-mortem* examination of fatal cases of syphilis. It was subsequently opposed by Mercurialis (*De Medicina Practica*, (Lib. IV., p. 470. Francof., 1601.) Paracelsus also disputed this opinion, on the ground that he had frequently observed other organs affected with syphilis, but rarely the liver. The idea, however, that the liver is the peculiar seat of syphilis was still maintained in 1604, by Ranchin of Montpellier, and afterwards by J. Keil (*Bresl. Siles. Dissert. inaug. de lue venerea.* Marpurgi, 1614), and Jonston (*Idœa universæ Medicinæ Practicæ.* Ludg., 1655).

The question could not be subjected to a thorough discussion, until pathologico-anatomical facts had been carefully collected. Bonet (*Sepulchret*, Lib. IV., Sect. 9) collected the materials which had accumulated before his time, and mentions several cases, where the liver of syphilitic patients was found "scabie et pustulis veluti quibusdam affectum;" but he maintains, that such appearances are rare, and that the view expressed by authors, to the effect that syphilis depends upon a "soluta unitas hepatis," is opposed to anatomical facts. Morgagni (*De Sedibus et Causis Morborum.* Venet., 1762, Epist. LVIII., p. 369) opposes the view, in still more decided terms, inasmuch as he asserts, that he does not remember ever to have found the liver diseased, in the bodies of persons who had suffered from syphilis. Subsequently, however, Astruc, Van Swieten, and Portal (*Maladies du Foie*, p. 363) reported cases of syphilitic disease

of the liver, but without assigning to them that extreme importance, which, for so long a period, had been a subject of discussion among the earlier physicians. These observations had almost been forgotten, until in recent times, Ricord (*Clinique Iconograph. de l'Hôpit. des Vénériens*) described morbid changes of the heart, the lungs and the liver, which he compared to syphilitic tumors or nodes (*Gummigeschwülsten*).[1] Rayer also (*Traité des Maladies des Reins*, T. II., p. 486) observed alterations of the parenchyma of the liver going along with nephritis albuminosa, which, he believed, must be referred to the syphilitic dyscrasia. The work of Dittrich (*Prager Vierteljahrschrift*, 1849, Bd. I.) was of a much more comprehensive character; it contained the first description of the characteristic appearances of syphilitic disease of the liver, and gave an impetus to a series of new investigations, by means of which the existence of this condition has been established. Among these researches may be mentioned those of Gubler (*Gaz. Médic. de Paris*, 1852. No. 17, and *ibid.*, 1854), S. Wilks (*Transactions of the Pathological Society*, Vol. VIII.), Bristowe (*ibid.*, Vol. X.), Virchow (*Archiv f. Path. Anat.*, Bd. XV., S. 266), and many others. It is true, that some authors, such as Böhmer (*Zeitschrift für ration. Therapie*, 1853, s. 88), and Vidal (*Traité des Maladies Vénériennes*, Paris, 1859), have called in question the syphilitic nature of the hepatic affection. But even if we disregard the frequency with which the anatomical changes occur in conjunction with constitutional syphilis, their appearance, their mode of development and their retrograde metamorphosis, agree so completely with the undoubted products of syphilis in other organs, that the doubts expressed by these writers of necessity lose their value.

2. *Anatomical Description of Syphilitic Hepatitis.*

The syphilitic process in the liver manifests itself in three different forms:—1. as simple interstitial hepatitis and peri-hepatitis; 2. as hepatitis gummosa; and 3. as waxy, amyloid, or lardaceous degeneration of the liver. All three forms may be found in the same liver, or may exist independently. The last of the forms, which is also produced by other cachectic conditions of the system, will be considered separately hereafter; at present we shall merely refer to the two inflammatory forms.

In the bodies of individuals who have suffered from constitutional syphilis, white depressions, like cicatrices, of a folded or radiated form, are often found upon the outer surface of the liver, the capsule of which is smooth or granular (*warzig*) and is usually thickened and firmly adherent to the neighboring organs, more particularly to the diaphragm, and, in rarer instances, to the colon and stomach. These depressions are of most frequent occurrence on the convex, but are also met with on the concave, surface; they are sometimes solitary, and at other times so numerous, as to make the liver present an irregularly lobulated form; they are rarely observed in the substance of the hepatic tissue, without reaching the external surface. On closer examination, they are found to consist of

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[1] In a private letter received from Prof. Frerichs, Gummigeschwülsten is translated: "Tumores inflammatione syphilitica orti." The terms "fibroid nodules," and "fibrinous masses," are the appellations ordinarily applied to the deposits in the liver by English writers. (See Wedl's Pathological History, Syd. Soc. Trans., p. 432; Wilks' Path. Anat., p. 329; Trans. Path Soc., VIII., 240, X., 21.)—TRANSL.

fibrous tissue, extending from the thickened capsule more or less deeply into the interior of the gland, the secreting tissue of which is atrophied. The fibrous tissue, in most cases, is dense and tendinous, and contains but few vessels; more rarely it is found soft and penetrated by large and small blood-vessels, which may be injected.

The large branches of the portal vein, the hepatic veins and the bile-ducts, as a rule, remain uninvolved, especially when the cicatrix does not extend very deep into the interior of the gland; narrowing and obstruction of these parts, and the consequent derangements of the circulation and of the excretion of bile,—ascites and jaundice,—are rarely observed.

In the second form of hepatitis syphilitica (*hepatitis gummosa*), the tissue of the cicatrices just described is seen to contain whitish or yellowish nodules of a rounded form and dried appearance, which usually vary in size from a linseed to a bean, but may be as large as a walnut.[1]

The substance of the nodule may not be conspicuous, from its being permeated by fibrous bands. Under the microscope, it is found to consist of oil-globules, and granules, cells loaded with fat and fibres of connective tissue. It thus resembles in its structural characters, the nodes (*Gummiknoten*), which are met with in the subcutaneous areolar tissue, beneath the peritoneum, in the testicle, &c., in cases of constitutional syphilis.[2]

The hepatic tissue intervening between these cicatrices or nodules, is either normal in its character, or, as has more frequently been the result of my own observation, in a state of fatty degeneration; in many cases, and according to Virchow's experience, in most, the loss of substance is compensated for by a characteristic hypertrophy, resulting from enlargement of the lobules and of the hepatic cells.

There are still other forms of syphilitic hepatitis where the development of areolar tissue is widely extended throughout the gland, and gives rise to simple or granular induration. Cases of this nature have already been described in this work, under the title of Syphilitic Cirrhosis, and their peculiarities have been pointed out.[3] Such indurations may have an independent existence, or may be associated with waxy or amyloid infiltration.

In most cases of syphilitic hepatitis, the entire volume of the liver differs but little from that of the normal condition; in rare cases, it is considerably reduced, whilst, on the other hand, it is usually enlarged when there is co-existent amyloid degeneration. Of 17 cases, which have come under my own observation, the organ was reduced in size 4 times, and once it did not exceed twice the size of a man's fist; the volume was normal in 7 cases, and in 6 it was enlarged; in 5 of the last cases there was waxy degeneration.

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[1] See Frontispiece.

[2] These nodules have already been figured by Ricord, who correctly classified them with the tubercles of the areolar tissue observed in tertiary syphilis. Budd described them as encysted, nodulated tumors, the origin of which he referred to inflammation of the bile-ducts with inspissation of the secretion (*op. cit.*, p. 189). Oppolzer and Bochdalek (*Prager Vierteljahrschrift*, 1845. Bd. II. s. 59) regarded them as cured hepatic cancers, until Dittrich demonstrated their syphilitic nature. Virchow has traced most carefully the development and retrograde metamorphosis of the anatomical lesions produced by the syphilitic virus.

[3] See p. 102. Defour (*Bullet. de la Société Anat. de Paris*, 1851, p. 139), recorded, in 1851, a case of cirrhosis of the liver occurring in the body of a syphilitic patient; and Böhmer also speaks of large scirrhus-like masses of connective tissue, as existing in the livers of children affected with constitutional syphilis.

The form of the liver may be altered in various ways. In one case the left lobe of the liver was shrivelled up into an appendage, scarcely an inch in diameter (See Fig. 6, p. 106); in another case, the right lobe was reduced to one-half of its normal size, while the left was so greatly hypertrophied, both in length and thickness, that the organ presented a quadrangular form. Still more frequently, the outer surface is found divided by means of fissures into irregular lobules, some of which may project in the form of rounded tumors, and occasion errors in diagnosis.[1]

In addition to the hepatic lesions, we usually meet with remains of syphilitic disease in other parts of the body, such as cicatrices on the genital organs and in the pharynx and œsophagus, enlarged glands, cutaneous eruptions and tumefactions of the bones; enlargement of the spleen, and degeneration of the kidneys, together with the signs of deep-seated cachexia, dropsy, &c., are likewise frequently present. Cases, however, are met with, where it is difficult to discover any certain proof of the existence of syphilis; Dittrich and Virchow mention instances of this nature, in which, with the exception of cicatrices in the pharynx, no proof of previous infection could be discovered. Still, these observations are of such an exceptional nature, that they cannot be fairly employed to throw doubts upon the relation between syphilis and the hepatic disease, which has just been described.

Another question more difficult of solution, is, to what stage of the venereal infection does syphilitic hepatitis pertain?—is it to be classified with the phenomena of the secondary or tertiary stage? The mere development of cicatrices may occur in the secondary stage as has been shown anatomically by Dittrich, and clinically by Gubler. No case has come under my own observation, which I have been able to refer with perfect certainty to this period. At all events, in most cases, the hepatic affection is found accompanied by other tertiary symptoms, and the more important pathological changes of the gland, the fibroid nodules (*die Gummiknoten*), and likewise the waxy infiltration, must, like the syphilitic affections of the other internal organs, properly be classified among the tertiary forms of syphilis. Whether other injurious influences co-operate with the syphilitic virus in exciting the hepatitis during life, is quite uncertain. The use of mercury has been blamed for this, as well as for other consequences of the syphilitic infection, but certainly without cause, inasmuch as cicatrices of the liver are met with in cases where not a grain of mercury has been taken. Virchow, from the situation of the cicatrices, is inclined to think that mechanical injuries, such as contusions, &c., cooperate precisely in the same way as Du Verney accounted for syphilitic disease of certain portions of the skeleton. The question, as to the cause of the localization of the dyscrasia, is one of great importance, but which cannot be decided by the materials as yet in our possession.

3. *Symptoms and Diagnosis.*

The effects upon the system of the simple and the gummy syphilitic hepatitis are, in general, not very striking. The principal portion of the glandular tissue usually continues quite capable of performing its func-

[1] See Observations recorded below.

tions; and occasionally the loss of substance is compensated for by hypertrophy. The cases are rare, where the larger branches of the blood-vessels or blie-ducts are obliterated, and where the derangements consequent upon such obstruction ensue. In those cases, however, where there is extensive induration or amyloid degeneration of the gland, all the consequences of cirrhosis or of waxy liver are usually developed.

The cachexia, which not unfrequently accompanies syphilitic hepatitis, is attributable to the disease of the spleen, the lymphatic glands, and particularly of the kidneys, rather than to the cicatrices in the liver.

The symptoms which accompany the disease during life are often so insignificant, that the development of the cicatrices escapes observation entirely, and they are found quite unexpectedly at the *post-mortem* examination. Cases, however, occur, where the symptoms are sufficiently marked to render a diagnosis possible. Among the most common of these symptoms, is pain in the hepatic region, which at one time is limited, and at another extends over the entire organ. The pain is usually of a dull, tight character, but sometimes is sufficiently acute to be the subject of great complaint.[1] Its duration may be protracted; one of my patients complained uninterruptedly for three months; in another, there were intermissions of a week, followed by exacerbations, which were accompanied by slight fever.

Another, but much rarer symptom, is jaundice; it is usually but slightly developed and of short duration. I have met with it in a case of peri-hepatitis syphilitica, where it disappeared with the cessation of the inflammation, and likewise in another case of amyloid degeneration and fibroid nodules (*Gummiknoten*) of the liver. In the latter case the autopsy showed that the cause of the jaundice was enlargement of the glands in the fissure of the liver. Lastly, in a third case, the jaundice was the result of obliteration of a large bile-duct by a cicatrix proceeding from the concave surface. The gland in this case was remarkably enlarged, and its outer surface was felt covered with rounded painful nodes, which for a long time caused the case to be regarded rather as an example of cancer of the liver. The autopsy displayed an indurated liver, cleft by cicatrices, and infiltrated with amyloid matter.

Where pain and jaundice are absent, the alteration in the form and volume of the gland may, under certain circumstances, when the organ is appreciable by palpation, apprise us of the existence of syphilitic cicatrices of the liver. But frequently this is not the case; many of the cicatrices are completely concealed by the ribs, and elude all means of diagnosis.

The symptoms just mentioned only justify the assumption of the existence of syphilitic hepatitis, when other unmistakable indications of constitutional syphilis are present, because all of them, the pain, jaundice, fissures, and altered form of the liver, may be developed from other causes.[2] It is sometimes a difficult matter to avoid confounding it with cancer of the liver, inasmuch as the main characters of the latter disease, the painful, nodulated, hard tumors in the liver, may likewise exist in the syphilitic affection of the organ, when it is associated with waxy infiltration; in the cases where there is none of this infiltration, the prominences are much

[1] I had under my care a patient, who had been obliged to discontinue using the springs at Aix-la-Chapelle, because the pains resulting from syphilitic hepatitis were found intolerable.

[2] See Observation XXXV., p. 111.

softer than those of cancer. The existence of constitutional syphilis, the (mostly) temporary pain and tenderness, the enlargement of the spleen, and the frequently coexisting albuminuria, may lead to a correct diagnosis of the syphilitic form of disease.

4. *Treatment.*

Therapeutic interference is rarely necessary; rest, local abstractions of blood, warm cataplasms, saline purgatives, and, subsequently, Iodide of Potassium, suffice for the removal of the symptoms. In all cases, however, even after the cessation of the distressing symptoms, recourse must be had to a radical, antisyphilitic treatment, in order to prevent ulterior consequences. Where the hepatitis is accompanied by waxy infiltration, the proper treatment for the latter affection is to be carried out.

5. *Illustrative cases.*

The first case which I shall record, by way of illustration of the statements just made, has reference to a patient whose liver had become deformed in consequence of syphilitic hepatitis, which had existed for a long period without inflicting great injury upon the constitution, and in which there could be no doubt as to the diagnosis.

Observation No. XXXVII. (*bis*).

Chronic Bronchial Catarrh.—Ozæna Syphilitica.—Cicatrices on the Velum Palati.—Liver covered with deep fissures and nodulated projections, and, at some places, painful.

Susanne Kiesewetter, wife of a day-laborer, aged 55, was admitted into the Medical Clinique at Breslau, on the 11th November, 1855, and discharged on the 28th December. For four years she had suffered from cough, with mucous, but never bloody, expectoration. Eight weeks before her admission she had been attacked with Asiatic cholera, from which she had recovered, so that she had been able to leave the Cholera Hospital eight days before. Since then her habitual catarrh had greatly increased, and it was on account of this that she applied for advice.

On admission:—Form of chest normal; no dulness; sibilant and moist râles audible on both sides; no signs of emphysema; muco-purulent sputa. Four years before, the patient's nose had fallen in; a portion of the septum was destroyed; bloody, fœtid discharge from the nostrils; radiated cicatrices upon the velum palati and in the pharynx. Appetite unimpaired; stools of normal color and consistence.

The liver extended far below the margin of the ribs; it measured 17 centimètres (6¾ Eng. inches) in the mammary line, and could easily be felt through the thin, flabby abdominal walls. On palpation, rounded, smooth prominences were perceptible, varying in size from a walnut to a hen's egg, presenting the consistence of doughy, fatty, hepatic tissue, and divided by deep fissures. Here and there the organ was tender. There was no ascites and no jaundice.

The spleen was rather large and rounded.

The urine was dense, but free from albumen.

Under the use of Decoction of Senega, in combination with Muriate of Ammonia, and afterwards with Extract of Cinchona Bark and Extract of Myrrh, the respiratory spmptoms improved with tolerable rapidity. The patient afterwards took the Syrup of the Iodide of Iron for four weeks. The ozæna disappeared; the tenderness of the liver ceased; but the form and volume of this organ, as well as of the spleen, remained unchanged. Even three months after, when the patient again presented herself, there was no change in this respect.

The following case is an example of the gummy form (*der gummösen Form*) of syphilitic hepatitis, combined with induration of the liver and partial amyloid degeneration. The remarkable alteration in the form of the gland is interesting.

Observation No. XXXVIII.

Deranged Digestion.—Cachectic appearance and Debility.—Anasarca without Albuminuria.—Catarrh.—Liver enlarged, deformed, and tender upon pressure.—Splenic Tumefaction.—Death from Œdema of the Lungs.

Autopsy:—Cicatrices in the Pharynx and Œsophagus.—Catarrh of the Air-Tubes.—Remains of Peri-hepatitis and Hepatitis Gummosa, together with Circumscribed Amyloid Infiltration.—Firm Splenic Tumor.—Kidneys normal.

Caroline Richter, aged 67, a watchman's wife, was under treatment in the Medical Clinique at Breslau from the 18th to the 27th February, 1858. The skin of this woman presented a yellowish tint, and her complexion was pasty; two months before admission, her feet and hands began to swell, but, in other respects, she had never been ill. Her principal complaints were a failing appetite, great weakness, and palpitations. The heart's action was irregular, but there was no abnormal bruit; the cardiac dulness was not increased in extent, and the apex could be felt in the fifth intercostal space. The lungs were normal, with the exception of a fine crepitant râle at the right base; slight cough. The liver was enlarged, measuring in the mammary line 20 centimètres (nearly 8 English inches) and in the axillary line 16 (6¼ English inches); its margin extended below the lower border of the ribs as far as the umbilicus, and could be felt in this locality as a rounded, fissured tumor, which was tender upon pressure. Moderate enlargement of the spleen; bowels confined; urine scanty, but free from albumen. Was ordered to take Infusion of Rhubarb, with Liquor Ammoniaci Anisatus.[1]

On the 24th of February, the dropsy had considerably increased; the walls of the abdomen were œdematous, and fluid effusion could also be detected in the peritoneal cavity. Heart's action feeble and irregular; pulse 80 and small; extremities cold. The moist râles had extended over both lungs; there was frequent cough without expectoration; the urine was scanty, and contained traces of albumen. Wine, Liquor Ammoniaci

[1] See note, p. 92.

Anisatus, &c., were prescribed; but from this date, the patient became rapidly worse, and, on the 27th, death supervened under symptoms of œdema of the lungs.

Autopsy, 18 *hours after death.*

There was no important change in the cranium or its contents.

The mucous membrane of the pharynx and velum palati presented white, radiated cicatrices, and other cicatrices of a similar character were observed at the base of the epiglottis; the mucous membrane in these parts was injected and tumid, and that over the aryteno-epiglottidean ligaments was œdematous; the redness extended downwards into the bronchi. The lungs were congested and infiltrated with serum, with the exception of their margins, which were pale and dry.

Isolated ecchymoses were observed beneath the epicardium; the valves of the heart were normal; its muscular tissue was somewhat discolored and friable.

The liver extended about five inches beyond the ensiform cartilage, and its situation and form were peculiarly altered. The left lobe lay in the right hypochondrium, whilst the right was situated high up in the hollow of the diaphragm, and was entirely beyond reach of palpation. The length of the left lobe amounted to $5\frac{3}{4}$ inches, and of the right to 3 inches. The capsule of the left lobe was considerably thickened, and, at some places, covered with warty excrescences; the anterior margin was rounded, and here and there divided by superficial fissures. Numerous, deeply-penetrating cicatrices were observed on the atrophied right lobe, and several of these cicatrices contained grayish-yellow fibroid nodules (*Gummiknoten*). In addition to these deposits, there were tolerably large masses of uniform induration. The gall-bladder did not project beyond the margin of this lobe; it was firmly adherent, and contained a white calculus of cholesterine. The parenchyma of the left lobe contained patches, in which the cells were infiltrated with lardaceous matter (the addition of Solution of Iodine and Sulphuric Acid produced here the red, but not the violet color), and other patches, which appeared fatty and somewhat hypertrophied.

The spleen was rather enlarged, and contained, at the upper part, a grayish-red wedge-shaped deposit; at other parts it was dense, firm, and of a waxy lustre; but no amyloid matter could be detected in it.

The mucous membrane of the stomach and intestines was pale, and, at some places tumid, and œdematous.

The kidneys were congested; but in other respects unchanged.

The serous covering of the uterus was much thickened, and adhered to the surrounding organs.

There was a radiated white cicatrix at the entrance to the vagina.

The following observation seems worthy of notice, from the fact of the obliteration of many of the branches of the portal vein, and the consequent hæmorrhage from the intestinal mucous membrane, which, together with the advanced degeneration of the kidneys, hastened the unfavorable termination of the disease.

Observation No. XXXIX.

Persistent vomiting of Mucous Matter.—Œdema of the Feet.—Albuminuria.—Syphilitic Cicatrices upon the Forehead.—Indurated Chancre upon the Genital Organs.—Bloody Stools.—Death.

Autopsy:—Lobulation and Induration of the Liver from Syphilitic Cicatrices.—Obliteration of numerous branches of the Portal Vein.—Amyloid Degeneration of the Spleen (which was small), and of the Kidneys.—Hæmorrhage from the Mucous Membrane of the Small and Large Intestines.

Henriette Q., aged 45, the wife of a music-conductor, was in my Clinique at Breslau, from the 4th to the 14th of December. She stated that, for a year and a-half, she had repeatedly suffered from vomiting and swelling of the feet, and she had likewise had many attacks of erysipelas of the face.

The woman was pale and thin, and there was a broad white cicatrix upon her forehead, like that which results from a syphilitic eruption, and quite unlike the effects of erysipelas. The mucous membrane of the pharynx was dark-red, but free from cicatrices. The thoracic organs presented nothing abnormal. The patient's chief complaint was constant vomiting, coming on after every meal, often spontaneously, and associated with a clean tongue. The epigastrium was distended, and slightly tender upon pressure; the liver was of normal size and its margins sharp; the spleen was small. The bowels were confined and the evacuations were pale. The urine was scanty; it was loaded with albumen, and deposited abundance of renal casts containing fatty epithelium, and sometimes blood-corpuscles. There was an open, indurated chancre upon the labia minora, and the anus was surrounded by large condylomata.

The patient was ordered to take Lemon-juice.

On the 10th of December the urine was secreted in rather larger quantity, and was free from albumen; the vomiting was less frequent. There was no improvement, however, in the digestive powers, and the prostration was rapidly increasing. Pulse 66, and small; consciousness unimpaired; slight headache; no derangement of vision.

Seltzer Water with Rhenish Wine was prescribed.

On the 12th of December, the patient had frequent, dark-brown, bloody stools. There was persistent vomiting of a watery, mucous, pale-yellow fluid. Extremities cold. Pulse 70, and scarcely perceptible. Urine very scanty.

Was ordered to take Phosphoric Acid with Spirit of Nitric Ether.

The hæmorrhage from the bowels continued; the prostration rapidly increased, and death took place on December 14th.

Autopsy, 24 hours after death.

There was no important alteration in the cranium or its contents.

The mucous membrane of the bronchial tubes was slightly injected and covered with frothy mucus. The lungs were everywhere crepitant; the left lung was infiltrated with serum, and the right was emphysematous.

The right side of the heart contained a quantity of loosely-coagulated blood; the muscular tissue was flabby; the valves, on both sides, were normal.

The spleen was small, dry, and firm; its cut surface was of a waxy lustre, and displayed amyloid deposits, which yielded a diffused bluish-violet color, on the addition of the ordinary reagents.

The liver was somewhat enlarged, and was divided by numerous fissures passing from the outer surface deep into the substance of the organ, into lobes and lobules, varying in size from a hen's egg to a hazel-nut. Dense connective tissue was found in the fissures, which at many places insinuated itself likewise between the lobules, and imparted a cirrhotic aspect to the parenchyma; it was only at isolated places that cells were observed infiltrated with amyloid matter of a red reaction with iodine; the cells for the most part were but loosely connected, and contained coloring-matter or oil. On slitting up the portal vein from the fissure of the liver, and tracing it into the substance of the gland, it was ascertained that a large number of its branches were obliterated; their walls were compressed by the connective tissue of numerous cicatrices, and their channel was blocked up, partly by firm thrombi, and partly by adhesion of the walls.

The mucous membrane of the stomach was injected and softened, but without any loss of substance. The lining membrane of the small intestine was of a reddish-brown color, much relaxed, and covered with bloody mucus; that of the colon had a brownish-black appearance, and was very tumid, especially over the folds. No ulcers or diphtheritic exudation could be observed anywhere.

The kidneys were very large; their cortical substance was hypertrophied, and white dendritic masses were distributed through it; the pyramids were congested. The cortical substance contained a quantity of amyloid matter, having a violent reaction.

The uterus was small and shrivelled. There was an elongated indurated syphilitic ulcer upon the left labium minus. At the sphincter ani there were several flattened hæmorrhoids beneath the mucous membrane of the rectum, with condylomata upon the adjoining cutis.

The last case to be mentioned gives a picture of advanced degeneration of the liver, spleen, and kidneys, and of the consequent cachectic state of the constitution; the *caries sicca* or *usura syphilitica* of the cranial bones was very characteristic.

Observation No. XL.

Syphilis many years before.—At a later period, symptoms of Pulmonary Consumption. —Albuminuria. —Diarrhœa.—Dropsy.—Death from Exhaustion.

Autopsy:—Syphilitic Caries of the Cranial Bones.—Thickening of the Dura Mater.—Cicatrices in the Pharynx.—Tubercles at the Apices of both Lungs.—Deformed Waxy Liver with Syphilitic Cicatrices.—Waxy Spleen and Waxy Kidneys.—Amyloid matter in the Mucous Membrane of the Small Intestine.

Fr. Gierschberg, aged 58, a comptroller, was a patient in the Clinique at Breslau, from the 8th to the 27th of February, 1858. Five weeks be-

fore admission, he had become dropsical; before this he had only suffered from respiratory complaints, and in other respects had always been healthy. The man on admission was pale and emaciated, and his legs were swollen as high up as the knees. Great dyspnœa; respirations 56; pulse 94. The apices of both lungs were condensed, and the left apex presented the signs of a vomica; there was abundant purulent expectoration. Sounds of heart healthy. Appetite slight; two or three thin, pale stools daily. There was no hepatic dulness in the epigastrium; in the mammary line it amounted to 14 centimètres (5½ Eng. inches), and in the axillary to only 10 (4 English inches). Several rounded, painless lobes could be felt below the ribs. The spleen was not enlarged. Urine scanty, depositing a yellow sediment and containing abundance of albumen.

Decoction of Calumba with Liquor Ammoniaci Anisatus[1] was prescribed, and, in the evening, an opiate.

On the 20th of February, the œdema had extended as high as the hips; the urine was very scanty; the diarrhœa had ceased two days before. The sputa were tenacious, and expectorated with difficulty. Pulse 110, and small; respirations 58.

Decoction of Senega, Extract of Cinchona Bark and Elixir Pectorale[2] were prescribed.

On the 24th February, the sputa were more copious and more easily expectorated. Tongue dry. Three fluid evacuations. Pulse 116; respirations 48. The urinary secretion amounted to about 6 ounces in twenty-four hours. Great collapse.

The collapse increased until the 27th, when death by exhaustion ensued.

Autopsy, 20 hours after death.

The skull-cap was long and narrow, and on the right parietal bone there was a depression 1½ inch long and 1 inch broad, having the form of a flattened cone, and presenting a rough, eroded appearance; its margin was raised and thickened. The pericranium at this part was firmly adherent, but at other places appeared normal.

At the corresponding portion of the vitreous table, there was an excavation about the size of a silver groschen (6¼ Eng. lines, or less than a sixpence) of a porous character, with which the dura mater was firmly connected by means of ragged processes penetrating into the foramina of the bone. The dura mater at this place was three lines in thickness, and round about were observed velvet-like osteophytes covering the inner surface of the skull, but likewise intimately united to the dura mater. The dura mater in the middle cranial fossa, as well as around the foramen magnum, was covered with a mould of bloody, fibrinous matter. The membranes and substance of the brain were in other respects normal, except at the part corresponding to the thickening of the dura mater, where the arachnoid appeared white, and the pia mater was firmly adherent to the cortical substance, which was here somewhat atrophied.

Radiated cicatrices were observed upon the uvula and in the pharynx.

Both lungs were firmly adherent, the left by means of a thick coria-

[1] See p. 92.

[2] The elixir pectorale is a cough linctus, containing a number of ingredients, such as benzoine, myrrh, anise, liquorice, gum-ammoniac, saffron, and the roots of the Inula Helenium and Iris florentina, with rectified spirit.—TRANSL.

ceous membrane. The apex of the left lung was indurated and infiltrated with yellow tubercles; in the apex of the right lung was a vomica the size of an apple. No change of importance was observed in the heart or pericardium.

The spleen was of normal size and firm; its cut surface was dry; the trabecular framework was increased, and at some places there were glistening deposits, exhibiting a feeble amyloid reaction.

The liver was larger than natural, and very deformed. Its left lobe was very small, measuring only 1¼ inch in its transverse diameter, whilst the right lobe was 5½ inches broad and 7 inches long. The organ was connected by firm adhesions to the diaphragm and colon. The convex surface and the anterior margin of the right lobe were subdivided into rounded, nodulated lobules, by deep cicatrices, intersecting one another at many places (Fig. 8); numerous fissures were also found upon the under surface. The parenchyma was, at some places, congested, and at others, of a waxy lustre, and firm. The hepatic cells, and likewise some of the vessels, presented distinctly the red amyloid reaction, but nowhere the violet tint.

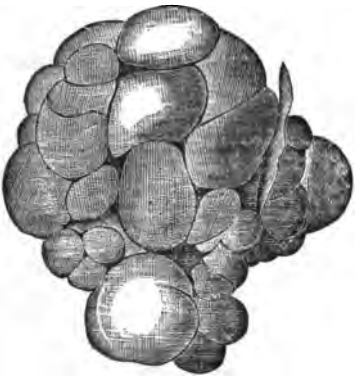

FIG. 8.—Deformity of the liver, resulting from numerous syphilitic cicatrices. The whole of the convex surface is subdivided into numerous rounded lobules. The left lobe is atrophied.

The mucous membrane of the stomach was pale. The follicles of Peyer's patches and of the solitary glands of the ileum were infiltrated with gelatinous matter; the villi and vascular loops presented distinctly the amyloid reaction, on the addition of Iodine. The mesenteric glands were unaltered. The lining membrane of the cæcum and colon was tumid and pale.

The kidneys were somewhat enlarged; their cortical substance was hypertrophied, and, at some places, infiltrated with a dense substance of a waxy lustre. The urinary bladder was normal.

The corona glandis presented an old radiated cicatrix.

CHAPTER IV.

THE WAXY, LARDACEOUS, OR AMYLOID DEGENERATION OF THE LIVER.

1. *Historical Account.*

THE earlier physicians had but an obscure conception of this form of disease, and the theories as to its nature have in the course of time passed through many different phases. Stahl and Boerhaave, however greatly their theories differed in details, both described this condition of the liver, together with many other forms of enlargement, under the titles of Infarctions, Obstructions, and Engorgements (*Anschoppungen*), and referred them to an accumulation of altered, thickened, or corrupted blood, within the blood-vessels. This doctrine of infarctions and obstructions necessarily gave way before the gradually improving notions as to the circulation of the blood and nutrition; it could not fail to be perceived, that obstruction of the vessels must give rise to atrophy, and not to enlargement of the gland, and hence it became necessary to transfer the stagnating mass to the exterior of the blood-vessels (Bordeu); at the same time, the term "engorgement" (*Anschoppung*) came into more general use.

Portal (*op. cit.*, p. 93) described a series of such engorgements of the liver, some of which, particularly the lymphatic form, belong to the disease under consideration. With the very imperfect chemical appliances at his disposal, he subdivided these engorgements into an albuminous, a gelatinous, and a mucous variety.[1]

It was not a step in progress when, at a later period, waxy degeneration was described as Hypertrophy of the Liver, by Andral (*Clinique Médicale*, T. IV., p. 354), Graves, Engel, and many others. Rokitansky (*Patholog. Anat.*, Bd. II.), was the first to give a clear account of the essential characters of lardaceous degeneration, and to recognize aright its pathogenetic relations to certain cachexiæ. Budd (*op. cit.*, p. 312), afterwards described the affection as "Scrofulous Enlargement of the Liver," whilst Oppolzer and Schrant (*Over de goed-en kwaadardige gezwellen*, Amsterdam, 1851), preferred the appellation of "colloid liver." Subsequent investigations have been mainly directed, on the one hand, to tracing the extension of the degeneration throughout the various tissues and organs of the body (the liver, spleen, lymphatic glands, kidneys, blood-vessels, mucous membranes, cartilage, nerve-substance, &c.[2]), and on the other, to

[1] Portal and others even make use of the term "lardaceous degeneration" (*Speckige Entartung*). Thus Portal observes (p. 365): "J'ai trouvé le foie excessivement volumineux, reduit en une substance pareille à du lard, soit pour sa couleur, soit pour sa consistance, dans une vieille femme, qui avait diverses exostoses et des ulcérations aux parties genitales."

[2] See S. Wilks, Cases of Lardaceous Tumors and some allied Affections, *Guy's Hosp. Reports*, 1856, Third Series, Vol. II.; also Virchow, *Arch. f. Path. Anat.*, Bd. VI., VIII., XI.; Friedreich, *ibid.*; and Beckmann, Bd. XIII., s. 94.*

* The author omits to mention the valuable communications made to the Physiological Society of Edinburgh in 1853, by Dr. W. T. Gairdner and Dr. W. Sanders, which were published in the *Edinburgh Monthly Journal of Medical Science* for February, 1854, p. 186; also another paper, by Dr. Gairdner, published in the same Journal for May, 1854.—TRANSL.

the determination of the essential nature of the waxy material, which is infiltrated throughout the tissues. Unfortunately, the researches in the latter direction have not as yet led to any satisfactory results. Virchow (*Archiv f. Path. Anat.*, Bd. VI., s. 135, 268 and 416), from the blue color produced by a Solution of Iodine, or by this reagent in conjunction with Sulphuric Acid, first observed by him in the corpora amylacea, came to the conclusion, that the substance must be classified with the vegetable carbo-hydrogens, cellulose, and starch, and named it accordingly "animal amyloid." Meckel, on the other hand, retains the appellation "lardaceous" or "cholesterine disease," and thinks that the essential character of the degeneration consists in the development of a certain fatty, or lardaceous matter, more particularly cholesterine. Against this last supposition, it has been justly contended, that the peculiar reaction remains unchanged, after removal of the fat, that it is not presented by other tissues, which contain abundance of cholesterine, and that, moreover, the color developed in the fatty matter of the bile, by the action of Solution of Iodine and Sulphuric Acid, differs from that of the so-called amyloid matter. The former view is certainly supported by the reaction; but all further proof from elementary analysis is wanting; and moreover, no one has yet succeeded in converting amyloid matter into any of the other carbo-hydrogens, and more especially into sugar, which can be easily effected in the case of vegetable starch and allied substances. Friedreich and Kekulé (*Archiv f. Pathol. Anat.*, Bd. XVI., s. 50) have ascertained, that the composition of the purest possible amyloid matter, obtained from the spleen, closely resembles that of the albuminous principles, and Schmidt (*Annal. d. Chemie und Pharmacie*, Bd. CX., s. 250) has arrived at similar results. It is obvious that the question is not yet settled and that the nature of the substance, which presents a reaction different from that of the albuminous principles, is still unknown; it may possibly be mixed with the albuminous principles in so small a quantity, that it is impossible to detect it by elementary analysis. Further investigations are necessary for the determination of its real nature.

2. *Anatomical Description.*

Waxy degeneration of the liver commences in the glandular cells, and the appearances which it presents in its early stages, are ill-defined and easily overlooked. The first indication observed is, that the middle portions of the lobules become reddish-yellow, translucent and firmer than natural, and sharply-defined from the surrounding dull-gray rim, and, as a result of this, the lobular structure is unusually distinct. On moistening the surface with Solution of Iodine, the glistening pellucid places are everywhere colored deep-red, the surrounding rim being only pale-yellow.[1] As the degeneration advances, the waxy, translucent appearance gradually extends over the entire area of the lobules, which become larger, while, after a time, their margins are ill-defined, and the parenchyma of the gland presents a uniform, smooth, yellowish-red, somewhat glistening surface, interrupted only by the patent orifices of the blood-vessels pouring out a

[1] The slighter grades of the degeneration can only be recognized by employing the iodine solution, which communicates a red color to each individual diseased cell and minute blood-vessel. Cases occur, where the degeneration is restricted to the branches of the hepatic artery; these diseased vessels would be easily overlooked, if they were not brought into view, in the form of red streaks or dots, by means of the iodine.

little thin blood. The portions of the gland affected in this way resemble fine sections of smoked salmon.

This morbid change of the glandular tissue occurs sometimes only in isolated, small, or large deposits,[1] but, most frequently, it is more or less uniformly distributed throughout the entire organ, although usually in such a manner that the disease is more marked in some places than in others.

In the slighter grades of the degeneration, the size of the gland is little altered, it may be normal or even reduced.[2] On the other hand, in the more advanced stages, the liver is considerably enlarged, and at the same time heavy; its capsule is smooth and tense; its consistence is of a peculiar doughy firmness; but its form remains unaltered. It is only where there is coexistent deposit of fat, that the margins are blunt and rounded. When granular induration is combined with the amyloid degeneration, the surface is covered with large and small nodules (*Höckerchen.*)

On tracing the progress of the degeneration in the individual cells, it is observed, that the finely-granular contents of the normal cells gradually disappear, to give place to a homogeneous, clear substance, which fills up the cavity of the cell. In a few solitary cells, the nucleus may still be observed, enlarged, and of a shining lustre, but in most of the cells the nucleus can no longer be detected, and the cell resembles a brilliant homogeneous scale. The cells, which are thus degenerated, are firmly connected to one another, and sometimes present large solid aggregate masses, in which neither cell wall, nor areolar matrix, can be distinguished.[3] (See Frontispiece, Fig. X.) In advanced degeneration, angular fragments are found here and there, which result from the destruction of the degenerated cells. (See Frontispiece, Fig. X.)

Changes, similar to those in the secreting elements of the gland are observed in the more delicate vessels, the walls of which become thickened, rigid, homogeneous, and lustrous, while their channel is narrowed and, not unfrequently, entirely obliterated. A vessel in this condition presents the appearance of a colorless cylinder, in which no trace of the more delicate structures can any longer be detected. It is a difficult matter to determine in every individual case, to which system the vessels thus affected belong; but so far, it is an ascertained fact that the disease is chiefly observed in the course of the ramifications of the hepatic artery; while the analogy of other tissues, such as the mucous membrane of the intestines and particularly the omentum, where the morbid process is more easily traced, favors the idea that it is the fine arteries which are first attacked. I have, however, repeatedly observed diseased capillaries, the locality of which appeared to correspond to the situation of the por-

[1] In one case, I have observed only three patches of deposit, with a diameter of from 1 to 2½ inches, distributed throughout the liver. These patches were sharply-defined from the surrounding somewhat fatty parenchyma. The largest of them was situated beneath the capsule in the convex portion of the right lobe, and penetrated about an inch into the interior.

[2] Out of 23 cases, I have observed 3 in which the size of the liver was normal, and 8 in which it was diminished.

[3] This circumstance would appear to have misled Budd into the view that the cells do not participate in the waxy degeneration. At p. 324, he observes:—"The foreign matter is not within the cells, but between them, and in advanced stages of the disease seems completely to take their place." This opinion is contradicted, in the most decisive manner, by the mode of development of the degeneration.

tal and hepatic veins. I have not met with any abnormal conditions of importance, in the larger branches of either vein; as a rule, injection succeeded tolerably well, whether the injected matter was thrown into the portal, or into the hepatic vein; but, in the case of the hepatic artery, the injection did not usually pass so far as the capillaries. Moreover, I have observed degenerated blood-vessels in the mucous membrane of the gall-bladder, and extensive ramifications of other vessels similarly affected in the capsule of the liver.

Whenever the hepatic cells, blood-vessels, or connective tissue are involved in the degenerative process, a deep-red color is observed, on moistening the surface with solution of Iodine, which, on the careful addition of sulphuric acid, gives place to a dirty-violet, or, more rarely, to a blue tint.[1]

The portions of the liver which remain exempt from the degeneration are distinguished, in most cases, by the greater amount of blood contained in them, as well as by their greater softness and moisture. The cells of these portions are easily separated from one another; they are of normal characters, and rarely contain much pigment, but more frequently those at the circumference of the lobules, and in exceptional cases the cells near the centres are in a state of fatty degeneration.

Waxy degeneration of the liver is tolerably often found combined with other forms of disease of the organ, and first and most commonly, with fatty deposit. I have observed this combination particularly, in cases of pulmonary tubercle. Here I have seen the lobules, and, indeed, whole series of them, on the surface of the gland and in the neighborhood of the larger branches of the portal vein, grayish white and fatty, whilst the deeper portions, and those surrounding the hepatic veins, presented a reddish translucent appearance and the firmness of the waxy liver. (Observation No. L.) Under such circumstances, the liver usually becomes very large, while its margins lose their sharpness and become rounded.

A second combination is that with cirrhotic induration. In this case, bands of connective tissue pass in various directions through the waxy parenchyma, which, in most cases, also contains a quantity of fat; the surface is nodulated; and the capsule is thickened, and at many places adherent. In this way is developed the large cirrhotic liver, which, in consistence and color, exhibits a certain resemblance to firm lard. (See Observation XXXIV.)

A third combination, of frequent occurrence, is that of the waxy liver with syphilitic cicatrices and fibroid nodules (*Gummiknoten*), which has been already described, and which is distinguished by the irregular division of the organ into lobes.

I have likewise, on one occasion, observed ramified, waxy degeneration in a simply atrophied liver, the remaining portion of the parenchyma of which contained shrivelled cells, some of which were loaded with fat, and others with pigment. Together with this condition of the liver, there was a waxy spleen and waxy kidneys, accompanied by great ascites and a moderate amount of anasarca. The case was that of a man aged 47, who had formerly suffered for a long period from muscular rheumatism, and who was afterwards admitted, with emphysema of the lungs and chronic albuminuria, into All Saints' Hospital at Breslau, where he

[1] On the whole, the blue reaction of the waxy substance is less frequently observed in the liver than in other parts of the body.

sank from exhaustion. Neither syphilitic disease of the bones, nor any of the other causes of waxy degeneration could be discovered.

Lastly, one other case of cirrhotic waxy liver, which was developed in the course of constitutional syphilis, appears to me worthy of notice. The patient was of a leucophlegmatic habit of body, had suffered repeatedly from attacks of epistaxis, and ultimately died from exhaustion. Ascites, albuminuria and great enlargement of the spleen were ascertained to exist during life. In addition to the cirrhosis and waxy degeneration, the liver was found to contain numerous gray nodules, the size of millet seeds, which consisted of rounded, nuclear formations and became dotted red when treated with solution of iodine. The blood, from the portal vein, and from the right side of the heart, contained a large number of colorless corpuscles.[1]

As regards the chemical characters of waxy liver, I have thought it necessary to ascertain, whether any relations subsist between the glycogenic material, which it is part of the normal function of the liver to elaborate, and the waxy substance, which is the result of a pathological process. The decision of the matter was the more necessary, inasmuch as both substances are colored by iodine and both have been regarded as carbo-hydrogens. The results of my investigations have been of a negative character. I have repeatedly examined the waxy liver for sugar, but have invariably failed to detect it. Moreover, no glycogenic substances could be discovered, which are not found in abundance in the healthy livers of animals by the same method of examination.[2]

Waxy degeneration is scarcely ever restricted to the liver; a similar form of degeneration is almost invariably observed in other organs, especially in the kidneys and spleen, and often likewise in the lymphatic glands and in the mucous membrane of the gastro-intestinal canal. In addition to this, we can usually at the same time discover the remains of chronic diseases of the bones, the indications of constitutional syphilis, tubercle, or cancer, &c.,—lesions, whose relation to waxy liver will be taken into consideration in discussing the etiology of the affection.

3. *Etiology.*

General circumstances.

Waxy degeneration of the liver is observed far more frequently in the male than in the female sex. Out of 68 cases (of which 23 were my own, and 45 the observations of others), 53 occurred in the male, and only 15 in the female sex,—a difference which is the more remarkable, from the circumstance that the diseases which usually predispose to the hepatic affection, by no means exhibit any predilection for males.

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[1] This case must be classified with Virchow and Böttcher's observations of leukæmic tumors in the liver. *Archiv f. path. Anatomie.* Bd. I., s. 569; Bd. V., s. 58; Bd. XIV., s. 483.

[2] The decoction of the waxy liver is distinguished from that of a liver containing glycogenic substances, by its paleness and clearness. Acetic acid produces a precipitate, which is again dissolved in an excess of the acid, in the former case, but not in the latter. In the decoction of the waxy liver, alcohol gives rise to a milky turbidity, and afterwards to a white, flaky sediment; but this sediment is perfectly soluble in caustic potash, is not, like glycogen, colored by iodine, and, when digested with saliva, furnishes no sugar.

Similar negative results were obtained from an examination of large quantities of the parenchyma of the liver and spleen, in a state of waxy degeneration.

As to age, the 68 cases were distributed in the following manner:—

Under 10 years	3	cases.
From 10 to 20 years	19	"
" 20 " 30 "	19	"
" 30 " 50 "	18	"
" 50 " 70 "	9	"
Total	68	"

It follows, that the disease, like scrofulous and tubercular affections, from which, under certain conditions, it results, is frequently developed during adolescence.

Waxy liver, almost without exception, makes its appearance in persons who have already become cachectic in consequence of other morbid processes, and whose nutrition has been seriously impaired. Among the pathological processes which predispose to the disease are the following:—

a. *Diseases of the Bones.*

Among these we particularly observe caries and necrosis, which commence on a scrofulous basis in the joints or vertebral column, or, more rarely, in the shafts of the long bones, and gradually spread. Those affections of the bones, however, which have a traumatic origin, or which supervene upon rheumatic periostitis or upon simple ulceration of the soft parts, such as ulcer of the leg, may likewise give rise to waxy liver, provided they have existed for a long period. It is frequently found that the disease of the bones has been cured long before the waxy degeneration of the internal organs begins to be developed. Rickets may also lead to the development of waxy liver.[1] (See Observations XLVI. and XLVII.)

b. *Constitutional Syphilis.*

This is one of the most common predisposing causes of waxy degeneration, not only when the osseous system is attacked, but even when the bones are quite uninvolved. Observations of this nature have already been collected by Portal; additional examples have since his time been recorded by Rayer, and, still more recently, by Rokitansky, Dittrich, H. Meckel, S. Wilks, Virchow, and many others. In this, as in almost all other diseases consecutive to syphilis, it is not the syphilitic virus, but the mercury which has been blamed; Graves and Budd, for example, refer the

[1] Glisson (*Anat. Hepat.*, p. 99) long ago observed: "Hepar in rachitide laborantibus prægrande esse." Bianchi likewise (*Histor. Hep.*, T. I., p. 130) remarks: "In enormem, naturalis tamen habitudinis, grandescit hepar in pueris rachitide affectis." Portal also (*Observ. sur la Nature et le Traitement du Rhachitisme*, pp. 29, 168, 170, &c.; *Maladies du Foie*, p. 414) states, that the liver of persons suffering from rickets is very large, and sometimes resembles lard in color and consistence. Rokitansky mentions rickets among the causes of lardaceous liver. My own experience on this point is not extensive. I have seen rickets complicated with waxy liver only once, but with fatty liver frequently. Lambl and Loeschner, of the Francis Joseph Children's Hospital (1860. p. 328), have described very carefully a case of rickets, complicated with amyloid degeneration of the coats of the intestine and of the spleen, and with fatty liver.

waxy liver to a combination of syphilis and mercurialism. Whether the mercury co-operates in any way, is a question to which it is difficult to give a decided answer. Although some English physicians speak very decidedly upon the point, I am not myself acquainted with any case where a swelling, and still less a decided degeneration of the liver, has resulted from the use of mercury. The cases which have been recorded as proofs, are all of such a nature, that it may be assumed that there were other co-operating causes. As regards the waxy liver of syphilitic persons, I know one instance where it was developed, although the syphilis had never been treated at all, owing to the circumstance of the primary ulcers healing spontaneously, and likewise another, where only a very short and superficial treatment was attempted, and where, at all events, there had been no abuse of mercury. Moreover, it appears from the investigations of Gubler (*Gaz. Méd.*, 1832), that waxy degeneration of the liver is found even in newly-born children affected with syphilis. I think, therefore, it may be assumed, that the use of mercury has been unjustly blamed as a cause of the waxy liver, precisely in the same manner as it was erroneously thought by Wells, Blackall, and Gregory, to give rise to diseases of the kidneys,—an opinion which Rayer long ago protested against. (See Observations No. XXX. and XXXI.; likewise Nos. XXXVIII., XL., XLI., XLII., XLIII., XLIV., and XLV.)

c. *The Cachexia of Intermittent Fever.*

This is mentioned by Rokitansky as a cause of waxy liver, while Budd observes that he has never met with an instance of the disease which could be attributed to such an origin. It is true, that waxy liver is not a frequent consequence of intermittent fever, but cases do occur where no other cause for the degeneration can be discovered. (See Observations Nos. XLVIII., and XLIX.)

d. *Tubercle of the Lungs and Intestines.*

Compared with the ordinary fatty degeneration, the affection of the liver in question is rarely observed to accompany tubercle of the lungs and intestines. Still, there are observations which point to a pathogenetic relation between tubercle and waxy liver. Meckel long ago came to this conclusion; Wilks has published three, and Friedreich two cases of this nature, and further on, I shall give the details of two cases in illustration, under Observations Nos. L. and LI. To these I shall add one other observation, where the hepatic disease was preceded by the cancerous cachexia.[1] (Observation No. LII.)

e. *Unknown Causes.*

The etiology of waxy degeneration of the liver and other organs is sometimes obscure; it is occasionally observed, without our being able to discover any precise cause. Wilks has described two cases of this nature as "simple lardaceous disease," and Observations Nos. XXV. and XXXIV. are additional illustrations.

[1] Budd mentions a case where waxy liver was developed, after protracted suppuration of the kidneys, induced by phosphatic calculi.

The first question, which here suggests itself, is in what way the morbid processes just mentioned give rise to the peculiar degeneration of the tissues under discussion? There are two possible modes which must be taken into consideration: the degeneration may be due to deposits from the blood, the waxy or amyloid matter, either as such, or as some primordial form, being generated in that fluid in consequence of the local disease of the bones, &c., or the waxy matter may be developed locally in the affected organ, by the transformation, from some cause or other, of albuminous matter previously deposited. Virchow, who has given this question careful consideration (*Cellular-pathologie*, s. 338, *Archiv. f. pathol. Anat.*, Bd. VIII., s. 368, Bd.XV., s. 234), is inclined to the former view, which he supports by the mode of development of the disease in affections of the bones, where the adjoining lymphatic glands are first implicated and the secreting organs, viz., the kidneys, the liver and the mucous membrane of the intestine are not involved until a later stage. The idea, also, that general causes, of the nature of dyscrasiæ, are here in operation, is favored by the circumstance that an entire series of organs in different parts of the body are affected, either simultaneously or in succession. Moreover, the hypothesis of a local development of the substance appears to me very doubtful, for the following reasons:—because the circumstances which precede the degenerative process vary greatly in their character; circumscribed deposits are not often observed; the progress of the disease varies greatly; the morbid matter itself does not always present the same characters, at one time exhibiting a pale-red, and at another a violet or blue, reaction; and, lastly, because its direct origin from fibrine has been demonstrated in certain cases.[1]

The sequence in which the different organs degenerate varies greatly. At one time, it is the liver, at another, the kidneys, and at another, the spleen, which is first and pre-eminently attacked, whilst the other organs either remain exempt or only present indications of commencing degeneration. It is rare to find several, or all, organs affected to the same extent. The reason of this is not apparent. The supposition that it depends upon the preceding diseases, from which the degeneration results, has not, in my opinion, been satisfactorily proved; we observe as a consequence of syphilis, of diseases of the bones, of intermittent fever, and of tubercular affections, sometimes one, and sometimes another organ primarily affected, whilst the remaining organs either follow, without any precise order, or remain for a long period exempt.[2]

4. *Clinical History and Symptoms.*

The consequences of waxy degeneration are always of great importance as regards the parts affected, because the organs and tissues, so far as they are implicated in the disease, lose their functions. The hepatic cells cease to take part in the formation of sugar and the secretion of bile; the blood-vessels, when they are implicated, lose their capability of transmitting fluids through their walls, and cease to furnish the materials ne-

[1] Friedreich found a substance, presenting an amyloid reaction, in the old fibrinous layer of the sac of a hæmatocele.

[2] The theory appears to hold good only in so far, as that it is the kidneys which are first attacked in most cases of caries and necrosis, and that, in intermittent fever, it is usually the spleen which is first affected.

cessary for nutrition and secretion. The injurious consequences are, on the whole, more marked, the farther the degeneration has extended throughout the organs which play an important part in the elaboration of blood and in nutrition, such as the spleen, the lymphatic glands, the mucous membrane of the stomach and intestinal canal, &c. Hence, we can understand how individuals with a waxy liver present, as a rule, a pale, cachectic appearance, and how symptoms of anæmia and hydræmia[1] manifest themselves at an early stage, and, the more so as the predisposing morbid processes, the ulceration of the bones, the tubercular affections, &c., have usually already produced an exhausting effect upon the constitution.

The clinical history of the disease naturally varies in individual cases, according to the cause which has excited the morbid process, and the direction in which it extends; it assumes one form in cases where the kidneys and the mucous membrane of the intestines are attacked at an early stage, and another, where the spleen and lymphatic glands are chiefly implicated. These differences depend upon the deranged functions of the individual organs.

In most cases, the liver is found enlarged, and the enlargement is uniform in every direction, so that the form of the organ is not essentially altered. The surface remains smooth, but the organ feels firmer and denser, and its margin is somewhat more rounded than is usually the case. The increase in volume may be very remarkable. In one of my cases the weight of the organ amounted to 5·6 kilogrammes (12⅓ lbs. avoird.); this, however, is not constant. In 23 observations, I found the liver enlarged 17 times, reduced in size 3 times, and of the ordinary size 3 times. In most cases, there is only abnormal tenderness in the hepatic region, to such a degree that the patients are merely annoyed by a feeling of fulness in the right hypochondrium; in rare cases, acute pains come on, in consequence of an attack of peri-hepatitis, which occasionally supervenes upon the syphilitic form of disease. It is only in exceptional cases that I have observed derangements in the excretion of bile. I have met with jaundice in 2 only out of my 23 cases, and in both of them the lymphatic glands in the fissure of the liver were found enlarged. The portal circulation also is not usually interfered with to any remarkable extent, inasmuch as it is the branches of the hepatic artery which are first affected. I have only observed ascites 8 times, and in 4 of the cases the effusion was the result of peritonitis. The peritoneal inflammation was, for the most part, very acute; and in none of the cases could it be attributed to any external cause, such as puncture of the abdomen.

Enlargement of the spleen is a common accompaniment of waxy degeneration of the liver; the organ often attains a very considerable size, and in most cases exhibits the peculiar characters of the waxy spleen. An increase in the volume of the spleen is, however, by no means of constant occurrence, and still less so is degeneration of its tissue. Of my 23 cases, the organ was enlarged 14 times, and in 10 of these 14 cases it was likewise in a state of waxy degeneration; in 9 cases, the spleen was of normal size, or diminished, and in 4 of these cases there were amyloid deposits.

The functions of the gastro-intestinal tact frequently suffer no remarkable derangement, excepting what results from the defective secretion of bile, which manifests itself by tympanites and an alternation of pale and

[1] I have observed leukæmic conditions of the blood in two cases (Observation XXXI. and p. 173.) Medical literature also contains several observations of leukæmia coexisting with waxy liver and waxy spleen.

dark stools. In many cases, on the other hand, the functions of the stomach, and still more frequently those of the intestines, undergo important changes; the appetite ceases, and vomiting, with a clean tongue, supervenes from time to time; diarrhœa, with pale, mucous stools, ensues, without any obvious cause, and persists obstinately for a long period, or returns from time to time, with short intermissions.[1] Under such circumstances, the mucous membrane of the stomach and intestine is usually found to all appearances but little altered; on closer examination, however, important anomalies present themselves, more particularly in the finer arterial twigs, the walls of which undergo waxy degeneration, and become glistening, rigid and thick, while occasionally their channel is blocked up. The substance of the villi is likewise frequently found infiltrated with waxy matter,[2] or they become atrophied and destroyed over extensive tracts of the bowel. Sometimes the destruction even extends to the tissue of the mucous membrane itself, and irregular ulcers are developed, which penetrate deeply into the submucous tissue, and on the edges of which disintegrating shreds of mucous membrane may be observed. I have repeatedly observed the capsules of Peyer's patches and of the solitary glands enlarged, and of a grayish-white color.[3]

It is obvious, that these lesions of the mucous membrane, by deranging digestion and absorption, as well as by the profuse secretion to which they give rise, must contribute greatly towards the development of anæmia.

Equally important, in this respect, is the change in the urinary secretion,—the albuminuria, which, in the majority of cases, accompanies waxy liver, and which usually depends upon waxy degeneration of the kidneys. I have observed the kidneys large and waxy in 23 cases; with diseased glomeruli, ten times; atrophied, twice; and in a state of hydro-nephrosis, once.

5. *Duration and Progress.*

The disease is always a lingering one; its duration is wont to extend over many months; and its commencement is usually overlooked. Once begun, the degenerative process in most cases advances uniformly to a fatal termination, which is almost always induced by exhaustion, under symptoms of general dropsy. Sometimes the disease terminates more rapidly by purulent peritonitis, or by dysentery, pneumonia,[4] œdema of the lungs, &c. Recovery rarely takes place; and in the case of far advanced degeneration, it is questionable whether the degenerated tissues can ever regain their normal functions. That recently infiltrated waxy matter may, under certain circumstances, be reabsorbed, appears to me to be proved beyond a doubt, by the observations, which I shall subsequently detail, and by similar cases already published, by Graves and Budd. The removal of the hepatic enlargement, however, does not always lead to re-

[1] Among 23 cases, I have observed persistent vomiting 6 times; but in 1 of the 6 cases there was cancer of the cardiac orifice, and in another, simple ulcer of the stomach; in 11 of the cases there was exhausting diarrhœa.

[2] Lambl (*op. cit.*) has traced most carefully the degeneration and destruction of the intestinal epithelium, and the degeneration of the substance of the villi, of the follicles of Lieberkuhn, and of the muscular coat.

[3] See Observations to be presently recorded; also H. Meckel, *op. ant. cit.*, p. 292; Virchow, *Archiv. f. path. Anat.*, Bd. IX.; Friedreich, *ibid.*, Bd. XI.; and Beckmann, *ibid.*, Bd. XIII., s. 94.

[4] I have seen one patient die from gangrene of the lungs.

covery. I have seen cases where the use of the mineral waters of Karlsbad[1] certainly effected a reduction in the size of the organ, but where, nevertheless, the cachexia continued to progress, and the fatal termination was not arrested.

6. *Diagnosis.*

The diagnosis is, on the whole, not difficult. The uniform enlargement of the gland, its increased consistence, associated with tumefaction of the spleen, and often likewise with albuminuria, and succeeding to caries, syphilis, tubercular affections, &c., are indications which guide us with tolerable certainty. It is easy to avoid confounding the disease with hyperæmic swelling, because the causes and the concomitant symptoms are entirely different. The same remark is applicable to the fatty liver, which feels softer on palpation, is rarely associated with splenic or renal disease, and, moreover, gives rise to few constitutional symptoms. There are forms of waxy liver, however, which it is impossible to recognize, because the size of the gland is not increased. In such cases, it is usually the splenic or renal affection which predominates, and the degenerative process can, in general, only be suspected, from the nature of the preceding diseases.

7. *The Prognosis.*

The prognosis is in most cases unfavorable; and the more so, the longer the disease has existed, and the farther the morbid process has advanced. When the kidneys and the mucous membrane of the intestines participate in the degeneration, the case invariably terminates in death.

8. *The Treatment.*

Treatment can only affect the progress of the hepatic disease in question, when its existence is recognized at an early stage. Hence, in individuals suffering from caries or necrosis, constitutional syphilis, intermittent fever, &c., the condition of the liver, spleen, and kidneys, should be carefully noted; in particular, we should endeavor to limit suppuration of the bones at an early stage, and, if necessary, by surgical interference; the sequelæ of constitutional syphilis ought to be removed by suitable treatment, before the symptoms of deep-seated cachexia manifest themselves, &c.

When an indurated swelling of the liver has already developed itself, in addition to the indications for treatment furnished by the primary disease, we must endeavor to remove the abnormal deposit, to arrest the progress of the degeneration, and to limit, as far as possible, its injurious effects upon the formation of blood and upon nutrition.

According to my experience, the former object is best attained, at all events in cases induced by syphilis, by the preparations of Iodine, the Iodide of Potassium and the Iodide of Iron. In one case of waxy degeneration of the liver, spleen and kidneys, resulting from necrosis of the femur and constitutional syphilis, the symptoms disappeared entirely

[1] For the mineral constituents of the springs of Karlsbad, see Vol. I., p. 88, note. —TRANSL.

under the protracted use of the Syrup of the Iodide of Iron. (Observation XLI.)

In another case, where the syphilitic virus was likewise the original cause, but where there was the complication of a series of courses of mercurial treatment, an equally favorable result was obtained by the baths of Aix-la-Chapelle,[1] in conjunction with Iodide of Potassium.

Graves has seen good results from the employment of the Iodide of Potassium, in combination with Blue Pill;[2] but his cases are not described with sufficient accuracy, to enable us to distinguish them from other enlargements of the liver.

With the preparations of Iodine, may be included the neutral Salts, such as Sal-ammoniac, the Carbonate, Sulphate and Phosphate of Soda, and likewise the Salts formed by the vegetable acids, to which the property of resolving enlargements of the glandular organs of the abdomen has long been attributed. Budd (*op. cit.*, p. 335), recommends particularly the employment of the Muriate of Ammonia, in doses of from 5 to 10 grains 3 times in the day. By means of this medicine, he succeeded in removing an enlargement of the liver and spleen, which had existed for nine months, which was accompanied by emaciation, pallor and irritative fever, and for which Mercury, Iodine and other remedies had been tried without any effect. The result of my own experience leads me to the opinion, that we must be careful in employing the neutral salts, as well as the mineral waters which contain them in large quantity, such as those of Karlsbad, Vichy, Marienbad and Kissingen;[3] because they are apt to give rise to exhausting diarrhœa, and to aggravate the cachexia. The springs of Karlsbad exercise an unmistakable influence over the nutrition of the liver, inasmuch, as they increase the secretion of the bile; and from the circumstance of their removing fatty deposits, it is possible, that they may alter the nutrition of the liver in the case of waxy degeneration, and so effect a reduction of the tumefaction of the gland. But the function of the gland is not always restored, even when the swelling is reduced. I have seen the cachexia continue to advance under such circumstances, to a state of exhaustion, although the mineral water was only prescribed in small quantities. The alkaline thermal springs of Ems,[4] as well as the sulphurous waters of Weilbach,[5] the influence of which upon the liver has been proved by Dr. Roth,[6] are less open to the same objection, and are on that account more deserving of recommendation.

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[1] The springs of Aix-la-Chapelle are thermal, sulphurous, and saline. Their temperature varies from 151° to 112° Fahr. A pint contains 36 grains of solid matter, and about a fourth of a cubic inch of sulphuretted hydrogen. The solid matter consists for the most part of chloride of sodium, with a little iodide and bromide of sodium.—TRANSL.

[2] Graves certainly recommended Iodide of Potassium, but considered mercury in any form injurious. See "Clinical Medicine," 2d Edition, 1848, Vol. I., pp. 451–2.—TRANSL.

[3] For the composition of these mineral springs, see Vol. I., p. 88, note.—TRANSL.

[4] For the composition of the springs of Ems, see Vol. I. p. 88.—TRANSL.

[5] Weilbach, in Nassau, is on the line of railway between Frankfurt and Mayence. The springs of Weilbach have a temperature of 57° Fahr., and are sulphurous. The water is said to contain three cubic inches of sulphuretted hydrogen in every 16 oz., and about one-fifth of its bulk of carbonic acid gas. Except for the carbonic acid, it resembles the springs of Gilsland and Moffat in our own country. The springs of Weilbach are chiefly frequented for hæmorrhoidal tumors and other hæmorrhagic affections.—TRANSL.

[6] Die Bedeutung des kalten Schwefelwassers zu Bad Weilbach. Wiesenbaden, 1854.

The bitter vegetable Extracts, such as the Extracts of Taraxacum, Chelidonium, &c., and likewise the recently prepared vegetable decoctions, do not merit the confidence reposed in them by the earlier physicians, who did not distinguish the hyperæmic and other swellings of the liver from the waxy degeneration. The same may be said of the Nitro-muriatic Acid; there are no accurate observations proving that by its use, either internally, or in the form of foot-baths, or general baths, the degeneration in question may be ameliorated or removed.

Still less can I confirm the favorable manner in which some modern physicians recommend the use of Cod-Liver Oil. I have seen cases of well-marked waxy liver developed during the continuous use of this remedy, which had been resorted to in the treatment of scrofulous caries and tubercle of the lungs. (See Observations XLVI., XLVII., L.)

The preparations of Iodine, the Sesquichloride of Iron and the mild alkaline medicines, together with the sulphurous mineral waters, are the remedies which ought to be chiefly employed in the treatment of waxy liver. The effects of these remedies must be assisted by such dietetic measures as are best calculated to improve the general nutrition, such as easily digested, nutritious, chiefly animal, food; good air, exercise, regulation of the cutaneous secretions by warm clothing, salt-water baths, &c., and proper attention to the intestinal and renal secretions. A sluggish state of the bowels is to be counteracted by Rhubarb, Choleate of Soda, Ox-gall, Aloes and similar remedies; and the diarrhœa, which is wont to supervene in the later stages of the malady, we must endeavor to keep in check by astringents, such as the Extract of Logwood, the Extract of Rhatany, the Aqueous Extract of Nux-vomica, Tannic Acid, Alum, the Liquor Ferri Sesquichloridi, &c., to which Opium must be added from time to time, when necessary.

The urine must be carefully examined, and the first appearance of albuminuria must be met by derivation from the skin, especially by means of warm baths, followed by the vegetable astringents. The anæmia and hydræmia, which, as a rule, make their appearance in the advanced stages of the disease, we must endeavor to combat as long as possible, by attention to diet and regimen and the preparations of steel.

9. *Illustrative Cases.*

I give here the details of a series of cases of Waxy Liver, arranged according to the etiological influences, which preceded the degeneration of the hepatic tissue.

A. Syphilitic Forms.

Observation No. XLI.

Necrosis of the Femur.—Repeated Syphilitic Infection.—Secondary Symptoms.—Several Courses of Mercurial Treatment.—Albuminuria.—Enlargement of the Spleen and Liver.—Anasarca.—Improvement under Iodine of Iron.—Relapse.—Aggravation of Symptoms in consequence of inappropriate Treatment.—Renewed use of Iodide of Iron.—Chalybeates and Warm Baths.—Cure.

Herr R. J., a young merchant, had suffered for a long time from necrosis of the thigh, and after repeated infection with syphilis, had been

seized with secondary symptoms, which had been treated with preparations of Mercury, apparently with success. Two years afterwards, he became pale and cachectic and had anasarca, and on more careful examination, albuminuria was detected, together with firm enlargement of the liver and spleen. Iodide of Iron, and subsequently the mineral waters of Pyrmont and Lactate of Iron, along with warm baths, effected a marked improvement in his state: the albumen in the urine diminished to a mere trace, the tumefactions of the liver and spleen were reduced, and the condition of the blood improved. The patient was sent to the South of France for the winter; and here the morbid process underwent a fresh aggravation in consequence of chills and errors in diet, assisted by the inappropriate treatment of a physician of the place, who applied blisters to the hepatic region and administered purgatives. The patient returned in spring, with general anasarca, albuminous urine and considerable enlargement of the liver and spleen. The liver in the mammary line measured 18 centimètres (7 Eng. in.), and the spleen extended about 7 centimètres (2¾ Eng. in.) beyond the border of the ribs. I repeated the Syrup of the Iodide of Iron, together with warm baths and a non-irritating tonic diet. The Iodide of Iron was continued for many months by the ordinary medical attendant, and then simple preparations of Steel were substituted. The anasarca soon disappeared completely, and the albumen in the urine diminished to a mere trace. After an interval of seven months, I ascertained that the hepatic dulness in the mammary line measured only 10 centimètres (less than 4 inches), and the lower margin of the spleen had receded 4 centimètres (1½ inch), behind the ribs. The patient's nutrition, general appearance and strength had become satisfactory.

Observation No. XLII.

Secondary Syphilis.—Abuse of Mercury.—Pseudo-rheumatic Pains.—Jaundice.—Tumefaction of the Liver and Spleen.—Cure by drinking, and bathing in, the Mineral Waters at Aix-la-Chapelle, together with Iodide of Potassium.

Herr J., a naval captain, from P., had undergone repeated courses of mercury, for the cure of various secondary syphilitic affections. He had used the Red Oxide of Mercury, Corrosive Sublimate, Mercurial inunctions and the Iodide of Mercury, in the most indiscriminate manner, and without any attention to diet. When the patient presented himself to me, he was suffering from pseudo-rheumatic pains; the ulcers in the pharynx had healed, but an obstinate attack of gastro-enteric catarrh had existed for many weeks, and jaundice had likewise made its appearance, together with painful swelling of the liver. The liver in the mammary line measured 16 centimètres, and on the sternal line 10 (6¼ and 4 Eng. inches); the spleen also was considerably enlarged. I sent the patient to Aix-la-Chapelle,[1] where Dr. Wetzlar succeeded in curing the gastro-enteric catarrh by means of Chalk mixture and Opium, and then directed him first, to have recourse to the Baths, and afterwards to take the Thermal springs in conjunction with Iodide of Potassium, internally. After this treatment had been continued for four weeks, the jaundice disappeared, the liver returned to its normal dimensions, whilst the pseudo-rheumatic pains were almost completely removed.

[1] See p. 182.

Observation No. XLIII.

Syphilitic Disease of the Bones.—Syphilitic Ulcers of the Mucous Membrane of the Nostrils.—Pains in the Larynx.—Impending Asphyxia.—Tracheotomy.—Death.

Autopsy:—Stricture of the Larynx.—Lardaceous Liver.—Enlarged Spleen.—Fatty Kidneys.

David Janitz, a baker, aged 53, a man of pale, cachectic habit of body, and flabby muscular tissue, was admitted into the Hospital on the 4th of July, 1853. Twenty years before, he had suffered from a chancre, and two years before from ulcers of the skin, which had left glistening, white, radiated, cicatrices. Both tibiæ were swollen and uneven, but were free from pain at the time of admission. Eight weeks before, cough had set in, together with pains in the larynx, increased by pressure. Swallowing was difficult and painful; the voice was hoarse; and gradually dyspnœa set in. The patient had been treated during four weeks in another department of the Hospital, with Conia and Nitrate of Silver, but without any benefit. Ulcers, covered with reddish-brown scabs, were observed in the nose. The pains in the larynx were of moderate severity; there was tenderness on pressure over the margins of the thyroid cartilage, but not over the trachea. Both surfaces of the epiglottis felt smooth; its margin was sharp. There was no derangement of the digestion.

No change could be detected in the lungs. The sputa were mucopurulent, and sometimes mixed with plugs of blood.

Iodide of Potassium was prescribed, together with the local application of solution of Nitrate of Silver to the larynx.

From the 24th, the blood disappeared from the sputa, and only mucopurulent masses were expectorated. The breathing, however, still continued difficult; there was complete aphonia; the pains in the larynx gradually ceased under the protracted use of the Nitrate of Silver solution.

On the 29th, there was a considerable increase of the dyspnœa, which was not relieved by poultices and the inhalation of steam. On the morning of the 30th, the patient was found in a cyanotic condition, breathing laboriously, somnolent, and incapable of being roused. Tracheotomy was performed at 6 A.M.; but although the respirations continued for two hours, the patient did not regain his consciousness, and at eight o'clock he died.

Autopsy, on August 1st.

The integuments over the skull were hyperæmic posteriorly. The longitudinal sinus contained dark fluid blood. There were about two ounces of serous fluid at the base of the brain. The arachnoid was opaque at many places. The substance of the brain and the choroid plexuses were congested.

The pericardium contained two drachms of serous fluid; the muscular tissue and valves of the heart were normal. The lungs were much distended.

The papillæ at the base of the tongue were greatly developed. There was nothing abnormal in the velum palati; the left tonsil was somewhat

enlarged; the mucous membrane of the œsophagus was pale. There was a moderate amount of atheroma in the coats of the descending aorta. The left lobe of the thyroid gland was somewhat enlarged. Viewed from above, the epiglottis and glottis were not thickened, and the aryteno-epiglottidean ligaments were not infiltrated. The glottis, however, was so narrow and rigid, as not to admit the point of the little finger. The ventricles of the larynx had disappeared, and were filled up by a dense mass of areolar tissue, two lines in thickness. Below this, ulcers were found on both sides, with smooth margins and glistening white bases; the surrounding mucous membrane was much injected. Anteriorly, between the ulcers on either side, was situated the wound resulting from the operation; and still farther down, the necrosed plate of the cricoid cartilage projected through the soft parts which had been divided. The surrounding areolar tissue was livid, thickened and softened.

The lining-membrane of the trachea was covered with brownish mucus, and moderately injected. Some of the bronchial glands were infiltrated with gelatinous matter.

The left lung, particularly along its margins, was very emphysematous, and the apex at its centre contained a small mass of obsolete tubercle.

The right lung was likewise emphysematous, and at its apex presented slight puckered cicatrices; posteriorly, and inferiorly, the lung was œdematous.

The capsule of the spleen was thickened, and covered with numerous cartilaginous deposits, the size of a linseed. The organ was moderately congested and of normal consistence. Its weight was 0·622 kilogramme (22 oz. avoird.); its length 7¼ inches; its breadth 5 inches; and its thickness 1½ inch.[1]

The surface of the liver was smooth; a yellowish-white cicatrix was observed upon the left lobe, which penetrated into the parenchyma to the depth of three lines. The parenchyma was firm, and the cut surface, glistening and pale-brown. Cells in a state of waxy degeneration were detected everywhere in the centre of the lobules, which were surrounded by pale fatty rims. Here and there were patches, where a large number of the lobules were uniformly degenerated. The weight of the liver was 1·49 kilogramme (52½ oz. avoird.); the right lobe measured transversely 6 inches, and from before backwards 6¾; the left lobe measured 4½ inches transversely, 6¼ inches from before backwards; and the thickness was 2⅜ inches.

The bile in the gall-bladder was of a deep-brown color, and contained a number of small blackish concretions. There were several enlarged lymphatic glands in the immediate vicinity of the cystic duct.

The mucous membrane of the stomach was tumid, and in the neighborhood of the pylorus was faintly injected.

The mucous membrane of the ileum and cæcum was normal. The large intestine contained solid brown fæces.

The left kidney presented a small recent extravasation of blood beneath its smooth capsule. A patch (*plaque*), the size of a two-groschen piece,[2] of a yellowish-brown color, but not penetrating into the parenchyma, was found beneath the capsule, at the apex of the right kidney. The epithelium cells of the uriniferous tubes were loaded with fat; the glomeruli were free from amyloid degeneration.

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[1] Paris inches. See Vol. I., p. 13, note.—TRANSL.

[2] A little larger than a sixpence.—TRANSL.

The urinary bladder was normal. The urine was pale and contained no albumen. The pancreas was shrivelled and somewhat congested.

Observation No. XLIV.

Hæmatemesis.—Distention and Tenderness of the Hepatic Region.—Jaundice.—Thin pale Stools.—Feeble action of the Heart.—Dyspnœa.—Sudden Death under Symptoms of Asphyxia.

Autopsy:—Infarctions of the Lungs.—Thrombi in the Pulmonary Artery.—Simple Ulcer and Cicatrices in the Stomach.—Waxy and Fatty Degeneration of the Liver.—Small Spleen.—Normal Kidneys.—Ulceration and Osteophytes of the Skull-cap.—Cicatrices in the Vagina.

Rosina Kannige, a joiner's widow, aged 63, was under treatment in the clinical department of All Saints' Hospital, from the 15th to the 25th of April, 1858. Two years before, she had an attack of cholera, followed by protracted diarrhœa. Three months after this, she vomited two quarts of blood, with subsequent alleviation of the symptoms of indigestion, under which she had previously labored; after this, she suffered occasionally from cramps in the stomach. Six weeks before her admission, she began to complain of swelling in the hepatic region, which was soon followed by jaundice. The patient was of a pasty complexion, and the body was covered with a thick layer of flabby, adipose tissue. She confessed to having been in the habit, more especially latterly, of drinking a great deal of brandy. There was a moderate degree of jaundice; the respiratory organs were unaffected; the pulse was 60 and small; the impulse of the heart was feeble, and the cardiac action irregular, but there was no abnormal bruit. Appetite slight; tongue clean; stools thin and pale. The liver in the mammary line measured 14 centimètres (5½ Eng. in.); its outer surface was uneven; its margin could be felt, through the thin abdominal walls, divided by several fissures; the organ was tender upon pressure. No splenic dulness could be made out. The urine contained a quantity of bile-pigment, but no albumen. Tincture of Rhubarb was prescribed.

April 18th. The jaundice has increased; urine brownish-black; stools pale; hepatic tenderness increased. Pulse 40, small and irregular; moderate ascites; œdema of the feet. Hydrochloric Acid with Chloric Ether was prescribed.

April 23d. In the same state, except that the pulse, which was soft, small, and scarcely perceptible, had risen to 50. Heart's sounds faintly audible, but free from abnormal bruit; no cough; some dyspnœa.

On the 25th of April, an hour after a meal, which the patient had eaten as usual, she suddenly complained of dyspnœa, became pale, fell back, and in five minutes was a corpse.

Autopsy, 18 *hours after death.*

The skull-cap was congested. On the left parietal bone, was an ulcerated patch, half an inch broad, and three-quarters of an inch in length, surrounded by osteophytes. The dura mater was somewhat thickened.

The pia mater, as well as the brain, was anæmic. The bones forming the base of the cranium were at some places much attenuated.

The mucous membrane of the bronchi was pale. The left lung, at its upper and back part, contained a grayish-red firm mass of infiltrated matter (*Infiltrat*), 1½ inch broad and 3 inches long, which consisted of extravasated blood undergoing discoloration; lower down, there was another patch, the size of a walnut, of more recent date, and of a reddish-brown color, and in the lower lobe were several others of a dark-red hue. Similar patches were found in the right lung, the apex of which also contained isolated tubercles. The pulmonary artery, as far as its finest ramifications, was filled with an arborescent coagulum, which, however, was nowhere firmly adherent to the wall of the vessel. The coats of the vessel were at some places in a state of fatty degeneration. The right cavities of the heart were filled partly with reddish-brown, and partly with yellow, coagula. The valves on both sides were normal. The muscular tissue was tolerably thick, but yellowish-red and soft, and, on microscopic examination, was found to be in an advanced state of fatty degeneration. The aorta was slightly atheromatous.

The liver was enlarged; there was a deep tight-lace depression running transversely across the right lobe, with thickening of the capsule, and here and there cicatrix-like depressions, and flat nodulated elevations. The consistence of the organ was firm; its cut surface was of a sulphur-yellow color and waxy lustre. When fine sections were treated with Solution of Iodine and Sulphuric Acid, a red tinge was observed at the parts corresponding to the centre of the lobules; no bluish color was anywhere developed; there was much fat in the vicinity of the minute subdivisions of the portal vein. On making fine sections and removing the fat by means of ether, the vascular network was unusually distinct, and the walls of the vessels were found to be thickened from lardaceous infiltration. On melting the fatty matter, the pigment which impregnated the hepatic tissue could be obtained in the form of numberless crystals of hæmatoidine. The examination of the organ for sugar yielded a negative result.

There was a simple ulcer, the size of a groschen (rather less than a sixpence), and several cicatrices in the mucous membrane of the stomach, which was somewhat injected. In the ileum were two small ulcers with tumid margins, which assumed a violet tint when treated with solution of Iodine and Sulphuric Acid.

The kidneys presented a jaundiced tint; but were in other respects normal. There were several suspicious cicatrices in the vagina.

Observation No. XLV.

Syphilitic Infection years before.—Epithelial Cancer of the Penis.—Amputation of the Penis.—Albuminuria.—Dropsy.—Right Pleurisy and Œdema of the Lungs of a threatening character.—Bloody Urine.—Diarrhœa.—Urine at first abundant, and afterwards scanty.—Gangrenous Erysipelas.—Death.

Autopsy:—Amyloid Degeneration of the Kidneys, Spleen and Liver.—Purulent effusion into the Cavity of the Pleura.—Cicatrices in the Pharynx.—Old Thrombus in the Left Renal Vein.

C. Schmidt, a laborer, aged 37, had suffered, for a long period, from the symptoms of secondary syphilis. On the 9th of September, 1858, he

was admitted into the medical Clinique at Breslau, with the symptoms of effusion into the right pleural cavity and general dropsy, having been operated on six weeks before for an epithelial cancer of the penis. The dulness extended as high as the second left rib. Over the left lung, a fine crepitant râle was everywhere audible. There was a moderate amount of ascites. The hepatic dulness in the mammary line amounted to six centimètres (2⅓ Eng. inches.) The dimensions of the spleen could not be ascertained, on account of the anasarca. The urine was scanty, opaque, brownish-black and loaded with blood and albumen. Decoction of Senega with Oxymel Scillæ was prescribed, with the object of diminishing the œdema of the lungs, which threatened to become serious.

On the 20th, the urine was passed in large quantity, amounting to three pounds (2¼ Eng. pints) in twenty-four hours; its color was paler. There was slight dyspnœa, and much sero-mucous expectoration. The appetite was good. The bowels were opened three times in the day, and the stools were semi-fluid.

On the 27th, the quantity of urine rose to four pounds (3 Eng. pints), with a specific gravity of 1015, and, on the 29th, to seven pounds (5¼ Eng. pints), with a specific gravity of 1016. On the 1st of October, it again fell to four pounds, with a specific gravity of 1020. Meanwhile, the pleuritic exudation had fallen to the fourth rib; the dyspnœa and cough had ceased, and the anasarca had diminished. Decoction of Cinchona, in combination with Bitartrate of Potash, was prescribed; and this treatment was continued for a long period.

Until the 12th of November, under an abundant secretion of urine, and copious fluid stools containing but little bile, the dropsical effusions continued to diminish; but from this date, the quantity of urine again decreased, and its color was of various dark shades, owing to the admixture of blood. The appetite fell off; the anasarca increased, and ultimately, gangrenous erysipelas made its appearance in the thigh and scrotum, and terminated in death on the 29th of November.

Autopsy, 18 *hours after death.*

The cranium and brain were normal. The right pleural sac contained two pounds (1½ Eng. pint) of purulent effusion. The left lung was œdematous, and the lower part of the right lung in a state of splenization. There was nothing abnormal in the heart. The pharynx contained white radiated cicatrices. The mucous membrane of the stomach was thickened, and, in the pyloric region, of a bluish tint. The mucous membrane of the small intestine was pale, and, at some places, rendered red by Iodine; that of the colon was livid and spongy.

The liver was somewhat enlarged and covered with isolated cicatrices. Its consistence was firm, and its surface on section pale-red and glistening. The cells in the centre of the lobules presented a distinct red reaction, when moistened with Solution of Iodine. The gall-bladder contained a little pale bile.

The spleen was considerably enlarged; its parenchyma was firm, brownish-red, and of a waxy lustre.

The kidneys were very large; their cortical substance was grayish-yellow, and marked by numerous dense cicatrices. The glomeruli were in an advanced stage of fatty degeneration. The left renal vein contained an old thrombus, softened in the centre. The mucous membrane of the

bladder was thickened and injected. There was a cicatrix on the foreskin.

B. Waxy Liver resulting from Diseases of the Bones.

Observation No. XLVI.

Carious Ulceration of the Hip-Joint and Necrosis of the Femur of many years' duration.—Enlargement of the Liver and Spleen.—Albuminuria.—General Dropsy.—Protracted Use of Cod-Liver Oil in large doses.

Autopsy:—Large Waxy Liver, with deposit of fat.—Waxy Spleen (Sago-Spleen, Sagomilz) and Waxy Kidneys.

M. N., a girl, aged 10, was under treatment in All Saints' Hospital, at Breslau, for several years, on account of scrofulous ulceration of the right hip-joint. The morbid process had extended far along the femur, and, on several occasions, sequestra had been removed. About a year before her death, in the middle of March, 1857, the liver and spleen were observed to be enlarged; there was albuminuria, and subsequently general dropsy supervened. Death took place under symptoms of œdema of the lungs. The child had taken large doses of Cod-Liver Oil almost uninterruptedly for several years; Steel, Iodide of Potassium and diuretics were afterwards called into requisition, to counteract the increasing dropsy.

The autopsy, in addition to extensive destruction of the right thigh and general dropsy, disclosed an advanced stage of waxy degeneration of the liver, spleen and kidneys.

The liver was very large; its surface smooth; its margins rounded; its consistence firm, and its cut surface anæmic and glistening. The secreting cells were, for the most part, in a state of waxy degeneration, but here and there, branched deposits of fat, accompanying the ramifications of the portal vein, could be recognized by their grayish-yellow color and greater softness. Here, cells could be detected containing numerous oil-globules, and, in a few instances, pigment molecules. These cells were easily separable from one another, whilst, on the other hand, in the larger waxy patches the cells were everywhere firmly adherent, and at the margins of these patches vessels could be distinguished, with thickened rigid walls, and some of them completely blocked up. These parts assumed an intense red hue, when treated with solution of Iodine; after the addition of Sulphuric Acid, the blue color was only developed in isolated, oval flakes; the remaining portion of the parenchyma presented only a violet appearance. The hepatic tissue contained no sugar, but abundance of leucine.

The spleen was enlarged to three times its normal size; its consistence was firm, and over its cut surface, which was pale-red and dry, numerous bodies resembling sago-grains (*Sago-körner*) becoming blue when treated with Iodine and Sulphuric Acid, were interspersed.

The kidneys were enlarged; their cortical substance was grayish-yellow, and sharply defined from the dark-red pyramids. The degenerated glomeruli could be recognized even with the naked eye; and, after the employment of Iodine, they became everywhere very distinct. The epithelium was at some places normal, but for the most part fatty.

Observation No. XLVII.

Rickets.—Tumefaction of the Spleen and Liver.—Death from Bronchitis and Lobular Pneumonia.

Autopsy: — Rachitic Disease of the Cranial Bones, the Ribs, and the Bones of the Legs. — Lobular Pneumonia. — Waxy Spleen. — Fatty Liver with Waxy Degeneration. — Enlargement of the Mesenteric Glands.

Theodor Becker, aged 21 months, was for a long time during the winter of 1851–2 a patient in the Polyclinique at Kiel, suffering from Rickets, more particularly of the bones of the legs, for which he was treated with Cod-Liver Oil, the Syrup of the Iodide of Iron and Lactate of Iron. The child was of a pale cachectic complexion; the spleen and liver were considerably enlarged; and there was a moderate amount of effusion in the abdominal cavity. About the middle of February, the little patient was seized with bronchitis and lobular pneumonia, which terminated fatally on the 21st.

In addition to the lesions of the cranial bones, ribs and bones of the legs, the bronchitis, and the lobular pneumonia, the autopsy disclosed enlargements of the liver and spleen, which were evidently due to waxy degeneration.

The liver was very large; its surface was smooth; and its consistence firm and doughy. Its surface on section was grayish-yellow, and displayed insulated patches, of a reddish, translucent, glistening appearance, which were distinguishable from the surrounding fatty parenchyma by their greater firmness. The bile was grayish-yellow, scanty and opaque.

The spleen was enlarged to three times its normal size, and of firm consistence. Its cut surface was pale-red and of a waxy lustre, and presented numerous gray, translucent, globular masses of the size of a millet-seed or upwards.

The mesenteric glands were much enlarged, without any distinct deposit. The mucous membrane of the stomach was of a rosy hue; that of the intestine was pale and relaxed and covered with grayish-yellow fæcal matter.

The kidneys were rather large, but in other respects presented no alteration of importance.

The reaction with Iodine and Sulphuric Acid was at that time unknown, so that the experiment was not tried.

c. Waxy Liver resulting from Intermittent Fever.

Observation No. XLVIII.

Persistent Intermittent Fever.— Uniform firm Enlargement of the Liver and Spleen. — Dissipated Habits. — Right Pneumonia. — Poisoning by Liquor Ammoniæ Fortis. — Pharyngitis. — Left Pneumonia. — Death.

Autopsy:— Waxy Degeneration of the Liver and Spleen.—Inflammatory Infiltration of both Lungs.

Robert Kabot, blacksmith, aged 39, was admitted into Hospital on the 18th of July, 1859, and died on the 26th. The patient had suffered from

intermittent fever for a year, with brief interruptions, and was addicted to brandy-drinking. Since the 13th of July, he had complained of pain in the right side, which had supervened after an attack of rigors, and was followed by cough and reddish expectoration. The pulse was 116, and there was great elevation of temperature; but the patient did not feel remarkably ill; he gave distinct answers when spoken to, and presented no obvious symptoms of incipient delirium tremens. Posteriorly, on the right side of the chest, there was dulness extending as high as the middle of the scapula, together with bronchial breathing, consonating râles, and rusty sputa; above this, and also in front, and on the left side, there was nothing abnormal. The spleen was large, and extended about 4 centimètres (1½ Eng. in.) beyond the margin of the ribs. The liver was likewise considerably enlarged; its dulness on percussion measured 10 centimètres close to the sternum, 16 in a line with the nipple, and 13 in the axilla (4, 6⅓, and 5⅓ Eng. inches). The margin of the gland was rounded; its surface smooth; and its consistence firm. Infusion of Digitalis was prescribed.

In the afternoon, there was profuse perspiration, without any abatement of the fever; the exudation had not extended. A quiet night. Slight dyspnœa.

On the 20th, there was again a profuse sweat, together with a tendency to somnolence; tongue much coated; stools pultaceous; pulse 112, full; respirations 28. In the afternoon, the patient took by mistake a table-spoonful of Liquor Ammoniæ Fortis, and although he instantly drank abundance of water, until vomiting resulted, and then a quantity of oil, he became immediately hoarse, and for twenty-four hours had much pain on swallowing. After leeching and cold applications, this pain ceased, but the hoarseness continued, though unattended by dyspnœa; the fever increased in intensity.

On the following morning (21st,) no redness could be discovered in the gullet; and there was no tenderness of the epigastrium. Bronchial breathing began to be heard below the scapula on the left side. Infusion of Digitalis with Mucilage of Gum Arabic was prescribed.

On the 22d, the entire lower lobe of the left lung was already hepatized; on the right side the consonating phenomena were still observable, the same as before. There was some pain upon pressure over the larynx, with increasing hoarseness. The expectoration was purulent and tinged with blood.

On the 24th, pulse 128; respirations 40. The expectoration was less copious. Decoction of Senega with Elixir Pectoralis[1] was prescribed.

On the 25th, pulse 136; respirations 48. The exudation had not extended. Cyanosis, delirium and a tendency to stupor supervened. Benzoic Acid was prescribed in combination with the Senega. In the evening, tracheal râles.

Death took place at 2 A.M. of the 26th.

Autopsy, at 9 p.m. of the 26th.

Skull-cap thick and congested; dura mater thickened; some firmly-coagulated blood in the longitudinal sinus; about an ounce and a-half of pale serum at the base of the cranium.

Arachnoid opaque; veins of the pia mater enlarged and tortuous. Brain-substance hyperæmic; choroid plexuses congested.

[1] See Page 92.

The mucous membrane of the pharynx pale; the aryteno-epiglottidean ligaments somewhat œdematous. Larynx intact; trachea and bronchi slightly injected; a large quantity of frothy fluid flowed out of the bronchi upon pressure.

The left lung was adherent at its lower part by recent fibrinous exudation, which extended as high as the upper lobe. The lower lobe, throughout its entire extent, was in a state of red hepatization; the upper lobe was very œdematous. The right lung was adherent like the left; its upper lobe was œdematous, and posteriorly, slightly hepatized; the middle lobe was anæmic and emphysematous; the lower lobe was infiltrated throughout, and in a state of gray hepatization.

The pericardium was opaque at some places; a fibrous patch was observed over the right ventricle. The right cavities of the heart contained a firm, and the left, a loose, coagulum. The valves were normal.

The tongue and pharynx were red and denuded of epithelium. The mucous membrane of the œsophagus, from the cricoid cartilage as far down as the cardiac orifice of the stomach was covered with strips of grayish-yellow exudation, which were easily peeled off. The intervening membrane was much injected, and at some places livid.

Stomach normal. No traces of the effects of the ammonia; but the mucous membrane was somewhat relaxed, and here and there ecchymosed.

Pancreas normal.

Mesenteric glands large, without any distinct infiltration; mesenteric veins not enlarged.

Mucous membrane of the ileum and cæcum intact.

The left lobe of the liver was in contact with the enlarged spleen. The liver projected 7 centimètres (2¾ Eng. inches) beyond the margin of the ninth rib. The capsule of the spleen was thickened; its parenchyma was grayish-brown, and of a shining lustre; its consistence, increased; and its dimensions enlarged; the length being 7½ inches, breadth 5 inches, thickness 1¼ inch, and weight 0·56 kilogramme (19¾ oz. avoird.).

The liver weighed 2·15 kilogrammes (4 lbs. 11½ oz. avoird.); its transverse diameter amounted to 10½ inches; from before backwards the right lobe measured 9½ inches. The capsule was white and opaque, and its margins rounded. Surface smooth, the parenchyma had a pale reddish-brown appearance, and presented a glistening surface on section, and a firm, lardaceous consistence. A large number of the cells were in a state of waxy degeneration, and were colored bright-red when treated with Iodine and Sulphuric Acid. This reaction was particularly marked at the centre of the lobules, but, at some places, it was uniform over large patches of the parenchyma.

The gall-bladder contained a small quantity of thin, pale, yellow bile.

Kidneys and urinary passages normal.

Observation No. XLIX.

Persistent Intermittent Fever.—Typhus.—Tumefaction of the upper region of the Abdomen.—Vomiting.—Diarrhœa.—Œdema of the Feet.—Large, smooth Liver, and enlarged Spleen.

Autopsy:—Waxy Degeneration of the Liver.—Great tumefaction of the Spleen.—Pneumonia ultima.

Robert Nowack, a journeyman miller, aged 26, was under treatment in

the Clinical department of All Saints' Hospital, from the 12th of May to the 4th of July.

Previous History.—Eight years before, the patient had suffered for nine months from quartan intermittent fever, which at first was neglected, but ultimately was arrested by medical treatment. After some months, he was attacked with a disease, which, from the symptoms, appeared to have been a severe form of typhus. During convalescence, he lost a large quantity of his hair. In the year 1854, Nowack was again seized with a severe acute disease, the nature of which he was unable accurately to describe. After this, he continued to enjoy perfect health for some years. He dated the commencement of his present illness from the spring of 1858. The first symptom was persistent cutting pains in the side, and soon his strength diminished to such an extent that he felt it necessary to give up his work. Almost at the same time, he observed a swelling in the right hypochondrium and epigastrium. This swelling continued to increase during this and the following year, and at the same time the patient gradually got weaker. In other respects, his complaints were slight, and were mainly referrible to derangements of the digestion, deficient appetite, and tenderness upon pressure in the epigastrium, particularly after eating. During the last year, there had been obstinate diarrhœa to the extent of three or four fluid stools in the day. For a long time, during the previous summer, he had suffered from severe attacks of colic, returning several times in the day, and lasting from half an hour to one hour. During the whole of this time, he had no return of the intermittent fever. He had never suffered from syphilis. Tubercle was hereditary in his family. Out of thirteen sisters, two only were alive; four died after the twentieth year of life of pulmonary affections; and his brother, who was still alive, was in the colliquative stage of phthisis. (The brother died at the beginning of June.)

State on Admission.—On the patient's admission on the 12th of May, 1860, he had a cachectic, pale complexion; no jaundice; chest and neck covered with pityriasis versicolor. No febrile symptoms; thoracic organs healthy. The epigastrium and right hypochondrium were very prominent, and the margins of the prominence were distinctly perceptible to the eye.

Corresponding to the region just mentioned, the liver could be felt very firm and smooth. Its rounded margin could be traced over its entire extent by means of palpation, as distinctly as the arch of the ribs. The size of the gland as determined by means of percussion and palpation, was as follows:—in the sternal line, it measured 14 centimètres (5½ Eng. inches); in the mammary line, or from the middle of the fifth rib to 8 centimètres below the arch of the ribs, 17 centimètres (6⅔ Eng. inches); in the axillary line, 15 centimètres (6 Eng. inches). The lower margin of the left lobe extended from the median line, and passing somewhat upwards in a slightly convex line from the left mammary line, disappeared beneath the arch of the ribs. The other boundaries of the left lobe could not be distinguished, owing to the great dulness in the splenic region. The splenic dulness commenced at the lower margin of the sixth rib, and passed outwards along with this rib to the axillary line, whence it stretched in a straight line to the vertebral column. On taking a deep inspiration, the upper margin of this dull space was displaced downwards to the extent of half an inch. The spleen did not project beyond the arch of the ribs. Examination of the remaining abdominal organs yielded nothing abnormal. The urine contained a very small quantity of albumen.

The subjective symptoms were the same as those already mentioned under the previous history. The debility of the patient was very remarkable; he spent the greater part of the day in bed, and a walk of even a few minutes greatly exhausted him. The diarrhœa had somewhat abated. For eight days, the patient has suffered from short attacks of rigors, followed by protracted heat. These attacks usually came on at midday; but, in other respects, had no definite type.

Progress.—The febrile attacks ceased after some days, under the use of Tincture of Chinoidine. Quinine with Iodide of Iron was now prescribed for the patient, who was transferred to the Polyclinique. This treatment was continued until the end of May, but without any benefit. About this time, violent paroxysms of cardialgia, necessitating the use of narcotics, set in, and transient attacks of œdema of the feet made their appearance. The pains in the stomach abated, but did not entirely cease. The diarrhœa returned from time to time, but was not severe. At the middle of June, vomiting supervened without any obvious cause. This symptom returned about three or four times in the day, for the most part a few hours after eating; it exhausted the patient to a great degree, and obstinately resisted the remedies employed to counteract it—such as Opium, Morphia, Bitter Almond Water, Effervescing Powders, &c., until, at the end of eight days, after a trial of the Tincture of Iodine, it ceased. On one occasion, a small quantity of blood was found among the vomited matters. Two days afterwards, the patient lost a whole plateful of blood from the mucous membrane of the nostrils. On the 25th, he again felt tolerably well, with the exception of great bodily weakness, and the Iodide of Iron was repeated; but, on the following day, the nausea and frequent attacks of vomiting returned. The vomiting resisted all treatment, while at the same time the patient suffered from severe pains in the epigastrium and restless nights. From this date, the collapse rapidly increased, and œdema once more made its appearance in the feet. The patient continued in this state until the 2nd of July, when he became suddenly worse. In the evening only a few isolated words (in reply to repeated questions) could with difficulty be obtained from him; immediately after answering, he relapsed into a state of drowsiness. The collapse increased; and early on the morning of July 4th, death ensued, without any change in the symptoms deserving of notice.

Autopsy, 12 hours after death.

The cranial cavity was not permitted to be opened.

Both lungs, but particularly the left, were adherent, but the adhesions were easily torn. The lower lobes of both organs contained a soft, brownish-red pneumonic infiltration. On section of these portions, a quantity of bloody serous fluid escaped. A few old cicatrices and small dilatations of the bronchi were observed at both apices, but no recent tubercles.

The heart wall small and very flabby, and contained but little blood; its valves were normal.

The liver filled up the epigastrium, the right hypochondrium, and likewise the greater part of the left hypochondrium. The left lobe was only one inch distant from the axillary line. The distended stomach and intestines were compressed downwards to a great extent. The liver was everywhere adherent to the adjoining organs, the abdominal wall, the stomach, the diaphragm, &c., the adhesions at some places being slight, and at others firm. Its measurements were as follows: the greatest thickness of

the right lobe was 4¾ inches; its greatest length, 8, and its greatest breadth 7 inches; the breadth of the left lobe was 4¼ inches; and thus the breadth of the entire organ was 11¼ inches. The capsule was much thickened and of a dull-white color; its margins were rounded; and its consistence remarkably increased. On section, patches several inches in breadth were observed at some places, which presented an almost perfectly homogeneous yellow appearance, closely resembling smoked salmon, whilst at other parts the lobular structure was very distinctly visible. The parenchyma was almost everywhere very anæmic, dense, and glistening; a flat depression remained after pressure with the finger, without any laceration of the tissue. The spleen was rounded, and in form resembled the flattened top of a mushroom. It was entirely concealed beneath the arch of the ribs; its length was 7½ inches, and its breadth, 6½ inches. The parenchyma was reddish-brown, of normal consistence, and, under the microscope, presented no change of any importance. The right kidney was adherent to the right lobe of the liver, and the left to the spleen; the left was of normal size, and the right, slightly smaller than natural. The mucous membrane of the stomach in the pyloric region was of a slaty hue; that of the small intestine was pale, whereas that of the large intestine was congested, and at some places livid.

D. Waxy Liver resulting from Phthisis Pulmonalis.

Observation No. L.

Chronic Tubercle of the Lungs and Intestine.—Treatment by Cod-Liver Oil.—Waxy and Fatty Degeneration of the Liver.—Commencing Degeneration of the Spleen.—Kidneys normal.

Mrs. Jenke, aged 21, had suffered for two years from the symptoms of chronic tubercle of the lungs, and had been treated almost uninterruptedly during this period with cod-liver oil. She died under symptoms of colliquative exhaustion.

The autopsy revealed circumscribed tubercular infiltration at the apices of both lungs, isolated ulcers in the intestines and slight ascites, together with far-advanced waxy degeneration of the liver.

The liver was considerably enlarged, and presented the smooth surface, and the rounded margins, as well as the pale-yellow color, of a fatty liver. On section, the fatty portion might be seen forming a layer with indented margins upon the outer surface only, and sharply defined from the portion of the gland, which was infiltrated with waxy matter. The latter constituted the greater bulk of the organ, and was distinguished by its pale-red color, and its great lustre, as well as by its dense, firm consistence. At some places, and more particularly upon the under surface, and in the lobus quadratus, the fatty layer was thicker. The fatty portion was everywhere more congested than the other part. The larger blood-vessels contained nothing but a little thin blood; dendritic layers of fatty liver were observed everywhere accompanying the ramifications of the portal vein, whilst the divisions of the hepatic vein were bounded by glandular substance in a state of waxy degeneration. The minute bile-ducts contained only a small quantity of tenacious mucus, presenting the yellow tint of fat. On microscopic examination the hepatic cells in the pale rims were found everywhere loaded with fat; the pale-red substance on the other hand, presented an almost uniform amyloid infiltration, fatty cells being only

observed here and there in the vicinity of the fine branches of the portal vein. The red tint characteristic of amyloid matter was produced by the addition of solution of Iodine and Sulphuric Acid, but the bluish color was only developed at a few points. Several small vessels, with their coats in a state of amyloid degeneration, were observed at the margins of fine sections. The liver did not contain a trace of sugar. The bile was scanty and pale.

The spleen was of normal size, somewhat dry and firm; it only contained isolated waxy deposits.

The kidneys were anæmic, but in other respects normal.

The liver was examined in order to ascertain the amount of its inorganic contents. A portion of the organ, dried at a temperature of 110° (110° cent.=230° Fahr.), was found to contain 7·4 per cent. of ash, whereof 3·4 per cent. consisted of alkaline salts, and 4 per cent. of earths, the phosphate and sulphate of lime, together with traces of carbonate of lime and magnesia.

In another case of waxy liver, resulting from constitutional syphilis, the organ was composed of 68·44 per cent. of solid matter, and 31·56 of water; of the solids 38 per cent. consisted of a fatty substance having an acid reaction, and containing but little cholesterine, and 30·44 per cent. of glandular tissue. This residue freed from the fat, yielded 15·5 per cent. of ash, of which only traces were soluble in water.

Observation No. LI.

Symptoms of Pulmonary and Laryngeal Phthisis.—Diarrhœa.—Large, firm tumefaction of the Liver.—Ascites.

Autopsy:—Tubercular Deposits in the Larynx, Trachea, Lungs and Intestine.—Very large Fatty Liver, with circumscribed Waxy Degeneration.—Soft Spleen, with isolated Waxy Deposits.—Fatty Kidneys.

Franz Hiltscher, a tailor, aged 38, was under treatment for several months for tubercular disease of the larynx, lungs, and intestines, which presented the usual symptoms, and only attracted attention in consequence of the large, firm tumefaction of the liver, and a considerable amount of ascites, without any œdema of the feet.

The autopsy disclosed extensive tubercular ulceration of the mucous membrane of the larynx and trachea. The apices of both lungs were infiltrated with gelatinous matter, and contained yellow tubercles, together with two cavities, the size of walnuts. There were numerous tubercular ulcers in the ileum, some of which had almost advanced to perforation. About five pounds (3¾ Eng. pints) of slightly turbid fluid were found in the peritoneal cavity.

The liver was enormously enlarged, firm, doughy, and of a grayish-yellow color. On making a section, the parenchyma was ascertained to be, for the most part, fatty, but also to contain, more particularly in the vicinity of the hepatic veins, insulated patches in a state of waxy degeneration. The waxy portions assumed a distinctly violet hue on the addition of the ordinary reagents. The fatty parts presented a faint, jaundiced tint.

The spleen was slightly enlarged and soft, and did not exhibit the or-

dinary character of a waxy spleen; it was found to contain, however, isolated, firm deposits, where the parenchyma, when treated with Iodine and Sulphuric Acid, assumed a blue color.

The kidneys were of normal size and flabby; the cortical substance was grayish-yellow and contained fatty epithelium; the glomeruli, however, were healthy.

To these two cases I annex the details of a third, where the affection was developed in consequence of cancer of the uterus.

Observation No. LII.

Hæmorrhages from the Vagina and Stomach.—Cancer of the Uterus and of the Cardiac orifice of the Stomach.—Tumefaction of the Spleen and Liver.—Death from Exhaustion.

Autopsy:—Cancer of the Uterus and of the Cardia.—Amyloid Degeneration of the Liver and Spleen.—Dilatation of the Calices of the Kidneys.—Renal Calculi.

Auguste Güttler, aged 41, a barber's widow, had suffered for a year and a-half from the ordinary symptoms of cancer of the uterus (pains in the uterine region, fœtid bloody discharge from the vagina, hæmorrhage, &c.). Three months before she came under observation vomiting had set in, the vomited matters consisting at first of mucus, and subsequently of a chocolate-brown substance. The existence of cancer of the cardia was ascertained by passing a probang, while a vaginal examination disclosed an extensive cancerous ulcer of the vaginal portion of the uterus.

Her symptoms were: œdema of the feet; a moderate amount of ascites; pale, waxy complexion and great emaciation; no appetite; constipation; moderate enlargement of the spleen. The urine contained only traces of albumen.

The patient, at the time of her admission into Hospital, was in a state of extreme exhaustion, and died a few days after. The treatment was purely symptomatic.

Autopsy on January 31st, 1858, 36 hours after death.

There was nothing abnormal in the cranium or its contents.

The lower lobe of the right lung was infiltrated with an aplastic (*faserstoffarmen*) exudation. The pleura at this part was covered with fibrinous flakes.

At the cardiac orifice of the stomach, there was a rounded cancerous ulcer, implicating the under surface of the left lobe of the liver.

The liver was large and firm. Its cut surface was reddish-brown, and of a waxy lustre. The entire organ was affected with amyloid degeneration; but some portions were firmer, and of a more shining lustre than others. Fine sections of the denser portions assumed a uniform red hue, on the addition of solution of Iodine; whilst in the softer portions, this color was only developed in dots. The violet reaction was nowhere observable. The gall-bladder contained numerous polyhedral concretions.

The spleen was double its natural size, firm, and at some places infil-

trated with a waxy, glistening substance, but at other places free. The colored reaction with iodine was only faintly developed.

The pelves of both kidneys were distended with turbid urine. The cortical substance was reddish-brown and firm, but was not colored by the addition of the iodine solution. At the base of the bladder, there was a superficial, ulcerated mass of cancer, which had spread from the vagina. The vaginal portion and the neck of the uterus were in a great measure destroyed; the lympathic glands of the pelvis were infiltrated with cancerous matter. The mucous membrane of the intestinal canal was pale, and covered with hard, gray fæcal matter.

Neither the previous history, nor the *post-mortem* appearances lent any support to the supposition of constitutional syphilis.

CHAPTER V.

HYPERTROPHY OF THE LIVER.

1. *Historical Account.*

THE term "hypertrophy of the liver" has been much abused, inasmuch as it has been applied to all sorts of increased volume of the gland, not accompanied by very obvious alterations of structure. Hyperæmias, fatty and waxy degenerations, and even morbid new formations, come under this designation in medical literature. Hence, the observations of the earlier writers upon this condition of the liver are not to be depended upon. The same remark is applicable to most of the more recent communications, where the organ has not been submitted to microscopic examination, which can alone determine the characters of true hypertrophy, and enable us to distinguish it from infiltrations and degenerations. We pass over, therefore, the observations made upon simple enlargements of the liver by Bartholin, Bonetus, Bianchi,[1] Morgagni,[2] Portal,[3] and others, and do not venture to take into consideration the cases recorded by Lobstein, Andral,[4] Abercrombie,[5] and Cruveilhier,[6] as examples of hypertrophy, because they are accompanied by no certain proofs that the structural characters of the organ were normal.

2. *Anatomical Description of true Hypertrophy of the Liver.*

By hypertrophy of the liver we understand an enlargement of that organ, accompanied by a simple increase in the size or the number of the secreting cells.

Comparatively few traces can be discovered of the nutritive changes constantly occurring in the normal liver. Structural forms, indicative, on the one hand, of the development or new formation of the glandular cells, or, on the other, of their disintegration, are so rarely met with, that one is forced to regard them as differing from the cells of other glands in being of a more persistent nature. Under certain circumstances, this condition undergoes a remarkable alteration; changes make their appear-

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[1] BIANCHI (*Historia Hepatis*, Tom. 1., p. 130) observes: "Varia sunt hepatis vitia, quibus a statu hoc viscus naturali alterari potest, absque læsione functionis suæ. Sic respectu ad magnitudinem, frequenter in inspsectionibus vastissimæ molis offenditur hepar, duplæ scilicit ultra naturalem, triplæ, quadruplæ, &c., *in culpabile tamen undique substantia contextum.*"

[2] MORGAGNI, Epist. XXXVI.

[3] PORTAL, *Maladies du Foie*, p. 29.

[4] ANDRAL, *Clinique Médicale*, Tom. II., p. 354.

[5] ABERCROMBIE, *Diseases of the Stomach*. German translation by Von der Busch, p. 432.

[6] CRUVEILHIER, *Anat. Path. Génér.*, Tom. III., p. 66.

ance, which are unquestionably indicative of an increased growth or of a rapid new formation of the elementary glandular structures. Cells are observed in the enlarged organ, which attain to twice or three times the normal size, and almost all of which contain two or three large sharply-defined nuclei, each provided with one or several vesicular nucleoli. These cells are easily separable from one another, and have an irregularly angular form; their contents are more or less granular, and occasionally include isolated oil-globules or pigment granules. The lobules of the gland are enlarged to an extent corresponding to the growth of the cells, and stand out distinctly from the cut surface.

In other cases, we observe small, rounded, pale cells, firmly-adherent to one another, with a large nucleus, and only slightly opaque cell-contents, and at the same time numerous free, round and oval, granular nuclei. These young cell-formations make up the greater portion of the hepatic parenchyma, or they are found in small quantity in conjunction with the first described cells, which contain two or three nuclei. When the small cells predominate, the outline of the lobules is not very distinct, and the cut surface of the organ is usually of a uniform reddish-brown color.

The volume of the liver, as a natural consequence of this increase in the size and number of the cells, undergoes considerable enlargement. The gland may attain to twice or three times its natural size, or upwards, without its form being essentially altered. Its consistence is at one time dense and firm, at another, soft and flabby; the quantity of blood contained in its vessels may be increased or diminished.

3. *Etiology.*

The circumstances, under which this increased growth or formation of the secreting cells of the liver takes place, are various. Of the proximate cause, we know equally as little as we do of the laws, which regulate the nutrition of the liver in its normal state. Persistent hyperæmia appears, under certain conditions, to favor the development of hypertrophy of the organ; it is not, however, of itself sufficient, inasmuch as it often lasts for weeks or months without affecting the nutrition. Hypertrophy of the liver is observed,—

1. *In Cases where one portion of the Gland is destroyed, in consequence of various exudation processes.*

In addition to the deep cicatrices which are frequently developed as the result of syphilitic hepatitis, or from obliteration of the branches of the portal vein or from any other cause, the hepatic parenchyma is frequently found hypertrophied and greatly swollen, and provided with enlarged cells and lobules, so as to compensate more or less completely for the loss of substance.

In consequence of this swelling, the cicatrices become deeper than they were originally, the deformity of the gland is increased, while, at the same time, derangements in the functions of the gland are prevented.

2. *In Diabetes mellitus.*

There are certain forms of diabetes, in which the anatomical lesion consists in an hypertrophy or increased formation of hepatic cells.

In the winter of 1849, I examined at Göttingen, the liver of a man, 44 years of age, who had died of diabetes mellitus and pulmonary tubercle with pneumothorax, and found it to present the following changes[1]:—The organ was considerably enlarged; its form was normal, and its outer surface smooth. Its parenchyma was much congested, of a uniform brownish-red color, and without any distinct indication of lobules; its consistence was dense and firm. The cells were intimately adherent, and unusually pale. Their form was rounded, and their size, small, measuring from $\frac{1}{100}$ to $\frac{1}{180}$ of a line. All of them contained a large shining nucleus, and only a small quantity of gray, or occasionally yellowish granules. In addition to the cells, numerous rounded nuclei, with nucleoli, were observed, and also young cells, with the cell wall closely applied to the nucleus.

The liver of a female, aged 37, who had suffered from diabetes, and who died from caries of the petrous bone and erysipelas of the face, presented similar characters.[2] The only difference observable was, that in addition to the young cells and nuclei, some of the hepatic cells were enlarged, while others were of normal characters. The hypertrophy of the gland was less remarkable; it measured 12½ inches transversely; from before backwards, the left lobe measured 3 inches, and the right, 5½ inches; the greatest thickness of the right lobe was 2½ inches.[3]

To these observations may be added, in the first place, those of Stockvis (*Bijdragen tot de kennis der zuikervorming in de lever*, 1856), who has carefully traced the appearances indicative of an accelerated cell-growth (viz., large cells, some of which contained several nuclei, young cells and free nuclei) in the moderately enlarged liver of a female, aged 30, who died of diabetes. An increase in the volume of the liver has been repeatedly observed after death from diabetes,[4] although unfortunately the minute structure of the gland has not been examined. Thus Bernard (*Leçons de Physiol. Expérimentale*, Tom. I., p. 416, Paris, 1855), found

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[1] The patient was a husbandman, named Ahrens, who had come from Holtensen, and who had been a long time under treatment in the Academical Hospital for diabetes mellitus. Ultimately tubercle was developed in the lungs, and death took place suddenly from pneumothorax. Eight days before death, the sugar disappeared from the urine, while its specific gravity, which had previously varied from 1030 to 1038, fell to 1023, and ultimately to 1010. The kidneys, as well as the liver, were hypertrophied, firm and congested; the uriniferous tubes at some places were considerably enlarged, their diameter varying from $\frac{1}{75}$ to $\frac{1}{70}$ of a line. It ought also to be mentioned, that the vesiculæ seminales contained a large quantity of gray fluid, with a number of spermatozoa and a translucent, ruby-red concretion, the size of a pea.

[2] Beate Pohl was a patient in the Clinique at Breslau from 18th July to 24th December, 1854. She passed saccharine urine, the quantity of which varied from 2500 to 3000 cubic centimètres (88 to 106 fluid ounces), and the specific gravity ranged between 1025 and 1027. Carbonate of soda, the Millsprings of Karlsbad (see Vol. I., p. 88.—TRANSL.), were all tried without any benefit, except that the derangements of vision disappeared under the use of the alkali. Three weeks before death, the patient was attacked with otitis and caries of the petrous portion and mastoid process of the temporal bone, which were followed by erysipelas and death by coma. The sinuses and substance of the brain were healthy, and the lungs contained no tubercle. The pancreas was atrophied, and at some parts in a state of fatty degeneration.

[3] In one other case of diabetes mellitus, which was under treatment in the Hospital of the Brothers of Charity at Breslau, I found small, pale cells in the liver. I neglected to make any careful note of the size of the gland, which, however, was not enlarged to any remarkable extent.

[4] Mead (*De Vipera*, p. 39) long ago observed: "Secti ex diabete mortui manifestum fecerunt ita rem esse. Semper inveni in hepate steâtomatosi aliquid, isti non dissimile visum materiæ, quæ sæpe in ictero per alvum dejicitur, sed consistentiæ durioria."

the liver congested, and very large in the body of a diabetic patient, who had died suddenly from pulmonary apoplexy; it weighed 2,500 grammes ($88\frac{1}{10}$ oz. avoird.) and contained more than double the absolute quantity of sugar in a normal liver, although the percentage was nearly equal to the natural percentage.

Hiller (*Preuss. Vereinszeitung*, 1843, s. 77) saw the liver and kidneys increased to three times their normal size, and the spleen double its ordinary size, in a case of diabetes.

But, as Griesinger has rightly observed (*Archiv. f. Phys. Heilk.*, 1859), the abnormal state of the nutrition of the liver is by no means a constant accompaniment of diabetes. Out of 64 cases collected by Griesinger, there was considerable enlargement of the liver only in 3, and a moderate degree of enlargement in 10. I have likewise met with cases myself, where neither the size of the gland nor the characters of the cellular elements afforded any proofs of an increased nutrition. This circumstance, however, does not detract from the value of the observations above detailed; they only confirm the view, that there are differences in the causes of diabetes, and that forms of the affection occur in which the liver is more actively implicated than in others. The accurate discrimination of these differences will no doubt ultimately be of service in treatment.

3. *In Leukæmia.*

In addition to the tumefactions of the spleen and lymphatic glands, in patients suffering from leukæmia, the liver is not unfrequently much enlarged. This enlargement, in most cases, is unattended by any alteration in the structure of the organ, although in rare cases, waxy, or cirrhotic degeneration is observed. Virchow,[1] J. H. Bennett,[2] J. Vogel,[3] Uhle,[4] De Pury,[5] Friedreich,[6] Böttcher[7] and others, have recorded a series of observations in proof of the occurrence of an altered nutrition of the liver, in conjunction with this abnormal condition of the blood. Excepting the observations of leukæmic blood coexisting with cirrhotic and waxy degeneration of the liver already detailed (See Observation No. XXXI. page 105 and also page 175) which do not properly belong to this category, I have only met with one case myself, illustrative of the point under discussion.[8]

In leukæmic patients, the liver, as well as the spleen and lymphatic glands, is found tolerably often hypertrophied (in 10 out of 18 cases, Bennett). Its weight increases to four, six, or even ten and twelve pounds. Its consistence is either normal, or soft and flabby (Bennett, Friedreich); but more frequently it is dense and firm (Uhle, Böttcher). The organ is, as a rule, anæmic; more rarely, hyperæmic. The lobules are usually large and prominent; the secreting cells are of large size; most of them contain

[1] Virchow, *Archiv. f. path. Anat.*, Bd. I., s. 569; Bd. V., s. 57. *Gesammelte Abhandl.*, s. 190.

[2] Bennett, *Leucocythæmia, or White-cell Blood*, &c. Edinb., 1852. *Clinical Lectures.* Edinb., 1858, p. 840.

[3] J. Vogel, *Archiv. f. path. Anat.*, Bd. III., s. 570.

[4] Uhle, *Ibid.*, Bd. V., s. 376.

[5] De Pury, *Ibid.*, Bd. VIII., s. 289.

[6] Friedreich, *Ibid.*, Bd., XII., s. 37.

[7] Böttcher, *Ibid.*, Bd. XIV., s. 483.

[8] This was the case of a lad 17 years of age, who was under treatment in the Breslau Clinique, for leukæmia, with hypertrophy of the spleen and liver.

several nuclei, and are filled with a quantity of fine, granular matter.[1] Under such circumstances, grayish-white nodules, the size of millet-seeds, composed of nuclear formations and young cells, enveloped in a delicate fibrous capsule, are found in the hypertrophied liver; similar formations are likewise met with in the kidneys. We shall consider these lymphatic new-formations (*lymphatische Neubildungen*) more carefully in the Chapter on Hepatic Tumors.

In this form of hypertrophy of the liver, the functions of the organ are usually impaired; the secretion of bile is diminished; and in the cases recorded by Friedreich it was so far arrested, that the intestinal contents were destitute of bile.

The importance of the hepatic affection in the development of the leukæmia must not be overlooked. The hypertrophy sometimes commences long before any alteration is observable in the blood;[2] at other times, but more rarely, it is consecutive to the change in the blood. Bennett and Uhle have shown, that the increase in the size of the liver may take place after the development of the leukæmia, and may then advance rapidly.[3]

From this it would appear, that the causes of the two conditions are not always the same. This much only is determined, that the unfavorable progress of the leukæmia is hastened by the implication of the liver, and that the treatment of this complication is equally unavailing, as that of the fundamental lesion has hitherto been.

4. *From residence in Hot Climates and in Malarious Districts.*

Physicians, who have had an opportunity of practising in hot climates, have observed, that, after a long residence in such countries, the liver is wont to increase in size, without any actual structural disease. Le Vacher (*Guide Médicale des Antilles*, p. 212) remarks:—"Il est peu d'habitants des colonies, qui ne soient affectés d'hypertrophie, ou de quelqu'état anormal de cet organe." Haspel (*Maladies de l'Algérie*, T. I., p. 230) observes:—"En général, après un séjour prolongé dans ce pays, il n'est pas rare de voir le foie acquérir, même dans un état sain, un volume beaucoup plus considérable que celui qu'il avait en France." Cambay (*De la Dyssenterie des Pays Chauds*, p. 527) frequently found the liver hypertrophied, in individuals who died of dysentery.

Similar observations have been made with regard to the malarious districts of the temperate zones. Statements of this nature must, however, be accepted with great caution, until we possess more certain proofs of an increased cell-growth in the enlarged liver, so as to avoid confounding the

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[1] Friedreich gives the following description of the cells:—The swelling of the liver was produced by an enlargement—which in some cases was enormous—of its cellular elements. The glandular cells had attained to double, or even three times, their normal dimensions. At the same time, their form was for the most part irregular; almost all of them contained two, and some of them three, sharply-defined round or oval nuclei, with comparatively large vesicle-shaped nucleoli, while the space between the nuclei and cell-wall presented a granular opacity.

[2] In Böttcher's case, the hepatic tumor was observed six years before death, and two years before the enlargement of the lymphatic glands.

[3] In Uhle's case, the hepatic dulness in the axillary line increased from 6 to 22 centimètres (from 2¼ to 8¾ Eng. inches), and in the mammary line from 7 to 18 centimètres (from 2¾ to 7 Eng. inches) between the 20th August and 14th October.

affection with chronic hyperæmias, or with fatty and waxy degeneration, which frequently occur in malarious districts.

I have several times had an opportunity at Breslau, of observing the liver unusually large, and furnished with hypertrophied lobules and large secreting cells, without any definite cause or functional derangement having been known to exist during life. The question is still involved in much obscurity, and must be cleared up by subsequent investigation.

For the relative sizes and weights, and the boundaries of the liver in its normal state, the reader is referred to the weights and measurements given in the first volume. (See Vol. I., p. 13.)

APPENDIX.

APPENDIX

OF OBSERVATIONS AND EXPERIMENTS IN SUPPORT OF THE STATEMENTS MADE IN THIS WORK.

No. I. (*Page* 15.) Tertian intermittent fever of three months' duration, ultimately with coma during the fits—Pneumonia passing on to consolidation—Sudden death.

Pigment-spleen and -liver—Induration of the lungs—The brain free from pigment.

No. II. (*Page* 16.) Febrile gastric catarrh — Vertigo — Convulsions—Coma—Return of consciousness—Parotid swelling—Albuminuria—Death from exhaustion.

Melanæmia—Accumulation of pigment in the spleen, liver, the gray matter of the brain and the kidneys.

No. III. (*Page* 17.) Quotidian fever—Enlargment of the Spleen—Coma—Death.

Melanæmia—Accumulation of pigment in the spleen, liver, kidneys, and cortical portion of the brain.

No. IV. (*Page* 18.) Fever of an undecided type—Delirium—Coma—Vomiting—Death on the fifteenth day. No albuminuria.

Pigment in the blood, the cortical portion of the brain, the liver, kidneys, and spleen—Spleen slightly enlarged.

No. V. (*Page* 19.) Intermittent fever with convulsions and loss of consciousness—Type irregular—No rigor stage—Recovery under treatment with bark.

No. VI. (*Page* 20.) Intermittent fever of an irregular type—Two paroxysms of forty-eight hours' duration—Furious delirium during the paroxysms—Cure.

No. VII. (*Page* 20.) Quotidian intermittent, with severe vertigo—*Febris vertiginosa* of Paccinotti—Cure by quinine.

No. VIII. (*Page* 21.) Quotidian intermittent of four weeks' duration—Diarrhœa—Albuminuria and hæmaturia—Sudden stupor—Convulsions—Death.

Accumulation of pigment in the spleen, liver, kidneys, and brain.

No. IX. (*Page* 22.) Continued fever with symptoms like those of typhus—Coma—Albuminous and bloody urine, containing clots loaded with black pigment—Right pneumonia—Abortion—Death on the sixteenth day.

Spleen and liver soft and loaded with pigment—Occlusion of the tubuli uriniferi by pigment.

No. X. (*Page* 23.) Quotidian intermittent fever—Intermittent albuminuria—Anasarca—Dysentery—Rapid cure by means of quinine and iron.

No. XI.—(*Page* 25.) Persistent and oft-recurring intermittent fever, ultimately of a quartan type — Copious albuminuria — Fibrinous

casts containing pigment in urine—Œdema—Rapidly supervening ascites—Tapping after the inefficacious employment of steel and purgatives—Return of the fever—Paroxysm of two days' duration—Death from exhaustion.

Pigment in the spleen—Occlusion of the hepatic capillaries.—Atrophy of the liver—Accumulation of pigment in the kidneys—Consecutive pneumonia.

No. XII. (*Page* 27.) Intermittent fever of a tertian and quotidian type, lasting for seven weeks—Intestinal catarrh—Hydræmia—Anasarca—Improvement under preparations of steel—Relapse—Rapid increase of the dropsy—Unconsciousness—Death.

Pigment in the spleen and liver, without any implication of the brain or kidneys.

No. XIII. (*Page* 28.) Slight dysentery—Albuminuria—Death from exhaustion—No cerebral disturbance.

Accumulation of pigment in the spleen, liver, brain, kidneys, and pancreas.

No. XIV. (*Page* 29.) Abdominal typhus—Repeatedly recurrent intermittent fever—Dysentery—Exhaustion—Death.

Spleen and liver loaded with pigment—Liver atrophied—Dysenteric ulceration of the large intestines.

No. XV. (*Page* 39.) Constriction of the left auriculo-ventricular opening of the heart—Incompetence of the tricuspid valves—Marked venous pulse—Hæmoptysis—Albuminuria—Mechanical hyperæmia of the liver, stomach, and large intestine.

No. XVI. (*Page* 40.) Constriction of the left auriculo-ventricular opening of the heart—Repeated attacks of œdema of the lungs—Albuminuria—Congestion of the liver from obstructed circulation—Hæmorrhagic erosions of the stomach—Intestinal mucous membrane pale and tumid.

No. XVII. (*Page* 51.) Tedious delivery—Symptoms of peritonitis—Jaundice—Vomiting of black flakes—Delirium—Death.

Purulent effusion in the peritoneal cavity—Hæmorrhagic softening of the liver, and extravasation of blood beneath the capsule.

II. EXPERIMENTS IN SUPPORT OF THE THEORY OF ICTERUS, ADVOCATED IN THIS WORK.

Injection of pure bile into the blood of living animals.

In order to ascertain the effects upon the various functions resulting from the absorption of a large quantity of bile into the blood, but particularly with the object of tracing what becomes of the bile in the blood,—whether it is transformed, or is excreted as bile, and, if transformed, what may be the nature of the changes it undergoes,—a series of experiments were undertaken, the results of which are here given. Fresh ox-bile,—from which the mucus had been removed by means of alcohol, and the whole of the coloring-matter, by animal charcoal,—was employed for injection. Two or three, and in some cases, four or five, grammes[1] of dried bile were dissolved in from thirty to forty-five grammes of distilled water, and filtered; the colorless or pale yellow fluid was then carefully

[1] One gramme=15.4325 troy grains.

injected into the jugular veins, or more rarely, into the veins of the thigh, a quantity of blood corresponding to that of the injected fluid, having previously been drawn off by the opening in the vein, so as to prevent the vascular system being over-distended. In some cases, the inspissated bile of the Prussian Pharmacopœia was employed in place of fresh bile. Dogs were the animals on which the experiments were always performed; in most instances they had been previously etherized to a slight degree; but by the time of the injection the action of the ether had ceased, and could no longer mask any derangements of the nervous system, which might make their appearance. After the injection, the animals were placed in a box constructed for the collection of the urine, and carefully watched. In most cases, however, the urine was poured directly into the utensil by compressing the bladder, so that the possibility of the admixture of any foreign matter was completely excluded. Some of the animals died under symptoms of violent dyspnœa (obstruction of the capillaries of the lungs), owing to the entrance of air into the lungs or to the too great consistence of the injected fluid; in other cases the urine passed into the box was soiled; twenty-nine of the experiments succeeded in such a way that their results could be made use of.[1]

1. *Effects of Bile introduced into the Blood upon the functions of the Nervous System.*

In no case did any remarkable derangements of the nervous functions follow the injection. As soon as the bile entered the blood, the animals usually appeared, by licking with the tongue, to experience a change of taste; but in no case were stupor, convulsions, retardation of the pulse, &c., observable; vomiting alone occurred repeatedly (perhaps in one-fourth of the experiments), and particularly in those cases where the inspissated ox-bile was employed; in some of these cases, also, the experiment was followed by some drowsiness. A short time after the injection, so little that was unusual could be observed in the behavior of the animals, that in one the operation was performed four times in different veins without being productive of any lasting injury.

2. *Changes in the urine.—Appearance of bile-pigment, &c.*

The character of the urine which was voided after the injection varied; sometimes it contained a larger or smaller quantity of coloring-matter, and at other times it contained none; the former was the case in 19, and the latter in 10, of the 29 experiments.

The urine containing coloring-matter was always passed in small quantity; it was of a greenish-brown color, became turbid upon cooling, and then appeared green, rapidly depositing flakes, which, under the microscope, presented a finely granular appearance. When collected upon a filter, this deposit formed a dark, grass-green layer, which dried readily, and which, when recently dried, exhibited the characteristic properties of the coloring-matter of bile, becoming decomposed by the action of impure nitric

[1] I omit here the experiments which I performed seven years ago at Göttingen and Kiel with filtered ox-gall; inasmuch as the changes in the urinary secretion were not investigated, and, consequently, they are of but limited value.

acid, or by a mixture of sulphuric and nitric acids, with a lively play of colors of green, blue, violet, and red. The flakes which were suspended in the urine presented unmistakably the same play of colors, although in a fainter degree. The reaction of the urine was, in most cases, neutral or alkaline, the removal of the flakes of coloring-matter was speedily followed by an abundant deposit of triple phosphates; the specific gravity of the urine varied from 1012 to 1019. In 17 of the 19 cases the urine was albuminous, and after the separation by filtration of the green flakes, its color was blood-red, apparently owing to dissolved blood-pigment; no blood-corpuscles could be detected in the sediment. In two cases mere traces only of albumen could be found, along with an abundance of coloring-matter. This altered urine was voided from four to twenty hours after the injection; in one case, in which there was obstinate retention of urine for forty-eight hours, the coloring-matter was very distinct; but in most cases the urine, excreted from twenty-four to forty-eight hours after the injection, had regained its normal characters.

3. *Chemical examination of the urine, passed within 24 hours of the injection of bile.*

a. Examination of the urine which contained coloring-matter.

In five of the nineteen cases, the urine was freed by filtration from the pigment flakes, evaporated to dryness, and the residue extracted by means of ether, and afterwards by alcohol. In no case did the ethereal extract contain an amount of coloring-matter deserving of mention, but only traces of a yellow fat. The alcoholic extract was colored brown, and did not usually exhibit the reaction of bile-pigment; in one case only, where the animal had died of dyspnœa some hours after the injection, did the greenish-brown coloring-matter obtained by means of alcohol, and which upon evaporation separated in the form of flakes, present the properties of bile-pigment. This alcoholic extract, when reduced to the consistence of syrup, exhibited the colored rings characteristic of the chromogenic ingredients of bile; and green, blue, violet, and red rings were observed to be formed upon the vessel by the action of atmospheric air during evaporation.

Pettenkofer's test with sugar and sulphuric acid failed to detect any of the biliary acids.

In four of the five cases, the alcoholic extract contained leucine in considerable quantity.

In three cases crystals were obtained, which from their characters, must be assumed to have been taurine, but their quantity was too small to admit of their being further examined.

In five cases the urine filtered from the flakes was treated with neutral and basic acetate of lead; the precipitate was dried, digested with alcohol and boiled. The alcoholic solution, which only took up small quantities of the compound of lead, was freed from lead by means of solution of sulphuretted hydrogen and concentrated. There remained a substance like myeline, and likewise leucine in the form of globules, composed of concentric layers, or of drusic masses made up of acicular crystals, together with isolated crystals of urea. The urea as well as the leucine appeared to be left, owing to the imperfect washing-out of the lead-compound. The mass was repeatedly re-crystallized, but no crystalline forms corre

sponding to those of the acids of the bile ever made their appearance, nor did Pettenkofer's test in any case yield a distinctly positive result.

In order to prove the certainty with which the biliary acids could be detected in the urine by treatment with lead, a small quantity of ox-bile freed from coloring-matter, was dissolved in healthy urine; the greater portion of it could be obtained again by precipitation with lead; and the alcoholic extract of this precipitate, when freed from lead, presented distinctly the characteristic reaction with Pettenkofer's test. We may therefore conclude, that in the above experiments the biliary acids were not excreted in an unchanged condition in the urine.

b. Examination of the urine, which was passed free from coloring-matter after the injection of bile into the blood.

Of the ten cases in which the urine was passed clear and pale, there was invariably a remarkable increase of the secretion; the quantity voided in twenty-four hours not unfrequently amounted to about 1000 cubic centimètres (35 fluid ounces). This urine was carefully examined in eight cases. The specific gravity varied from 1008 to 1012: on the addition of nitric acid, the pale-yellow color was not changed in any way, so as to remind one of bile-pigment; no albumen was precipitated. In order to isolate any biliary acids which might have passed over unchanged, the secretion voided within the first twenty-four hours was precipitated by neutral and basic acetate of lead; the precipitate was collected and dried, and digested and boiled in alcohol; and then the filtrate was freed from lead by means of solution of sulphuretted hydrogen, and condensed. The quantity of the compound of lead, soluble in alcohol, appeared to vary according to the longer or shorter time that the precipitate had been washed with distilled water; in the former case the quantity was small, in the latter it was large. The residuum remaining after the removal of the lead, and the evaporation of the spirit of wine, exhibited in five cases very beautiful crystals of leucine, and twice, where the washing had been only superficial, urea also; the so-called myeline, was usually seen to accompany these substances. When treated with sugar and sulphuric acid, in two cases only did the residuum yield the red color indicative of traces of the acid of bile; in the other cases, there was a negative result. In one instance the colorless spirituous solution, after being freed from lead, exhibited, shortly before complete desiccation over the water-bath, the color of the chromogen of bile; in this instance, too large a quantity of free sulphuric acid was not present; at least leucine was found undecomposed in the residuum.

In one other case, where the urine, which was free from both coloring-matter and albumen, was at once condensed, flakes of a green coloring-matter were formed during evaporation, which with nitric acid presented the same reaction as was observed in the spontaneous precipitate of the first nineteen cases. This appearance was not produced in two experiments which were subsequently made in the same way.

In many of the cases the urine was precipitated by lead, and after the removal of the excess of this metal, evaporated. Isolated globules of leucine could usually be found in the residue, which was loaded with urea, but sometimes they were searched for in vain in the mass of other crystals.

The following propositions result from the preceding series of experiments:—

1. The injection of large quantities of bile into the blood of living animals is followed by no important derangement in the vital functions.

2. After the injection into the blood-vessels of large quantities of bile deprived of its coloring-matter, pigment is in most cases excreted along with the urine, which exhibits most of the characteristic properties of bile-pigment.[1] This coloring-matter closely corresponds in its behavior, with the products, which may be formed artificially by the action of sulphuric acid upon the acids of the bile.

3. In rare cases, in place of the coloring-matter we find chromogen substances, which are not transformed into coloring-matter until the evaporation of the urine upon exposure to the air.

4. Unchanged biliary acids are not found along with the coloring-matter, but leucine is usually present.[2] Taurine and glycine cannot be detected with certainty.

5. The injection of bile is, in some cases, not followed either by the excretion of coloring-matter with the urine, or by the appearance of large quantities of unchanged bile.

The causes of this anomaly are unknown; the failure in the appearance of the pigment is accompanied by a profuse secretion of urine, whilst its presence is almost always associated with albuminuria, and often also with hæmaturia. The appearance of the coloring-matter appears to be favored by whatever interferes with the respiration; at all events, it was particularly abundant in one animal, which died asphyxiated some hours after the injection, as also in two other animals, which had oil introduced into their air-passages with the object of obstructing their breathing.

The theory of that form of jaundice, which occurs without any structural lesion of the liver or bile ducts, which has been already discussed (page 116), mainly rests upon these observations. It is obvious that defects still remain to be filled up before this view can be regarded as firmly established. The only point proved with certainty is the possibility of the transformation of the biliary acids into coloring-matter, and the dependence of this transformation upon certain conditions, which appear to consist principally in the functional activity of the respiratory organs and kidneys. When these conditions are more accurately determined, the individual causes of the metamorphosis of bile in the blood, and the connection of the various pigments with one another, must be better understood than hitherto has been the case.

III. *Experiments on the rapidity of the absorption of bile after ligature of the ductus choledochus.* See page 120.

IV. *Experiments on the influence of a fatty diet on the generation of fatty liver.* See Vol. III., page 44.

V. *Experiments on the injection of oil into the portal vein.* See Vol. III., page 44,—note.

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[1] The insolubility of the pigment constitutes an apparent difference. The coloring-matter, however, of the urine of jaundiced patients frequently exhibits the same character; in many cases, it can be completely separated from the fluid by filtration.

[2] Experiments, with the object of detecting leucine in decomposing bile, have failed for the most part in summer; but a large quantity of leucine, along with crystals of taurine, have been obtained from human bile which has been for a week in a freezing state during winter. The quantity of leucine which is obtained through precipitation of the biliary acids by the neutral and basic acetate of lead, appears much too considerable to be probably induced by the decomposition of the mucus of the bladder.

I.—Results of Examination of Individual Gall-stones.[1]

No. 1. *Frontispiece, Vol. III., Fig.* 9.[2]

Physical Characters.—A large gall-stone, which passed from the ductus choledochus into the intestine and caused death by ileus. Its form was cylindrical, and it measured a little more than 3 centimètres (1·18 Eng. inch) in its long diameter. One end of the cylinder appeared rounded, and presented a cracked, bark-like structure, of a brown color; the other end was broken off, and in the centre of the broken surface a nucleus 1·5 centimètre (7 Eng. lines) was observed. This nucleus was composed of brilliant, laminated masses of crystals, arranged in a radiated manner with tolerable firmness, and enclosing only a small quantity of yellowish-brown pigment. Its outer surface was slightly rough, and when crushed it broke down into wedge-shaped fragments.

Around the nucleus was a yellowish-brown, dried mass, composed of concentric layers, like the annual rings of a tree, without any distinct crystalline structure, and of a consistence resembling that of rotten wood. This mass on the broken surface of the concretion had a thickness of 0·7 to 0·8 centimètre (3½ to 3¾ Eng. lines), but in the long axis of the cylinder it was considerably thicker. It surrounded the nucleus like a shell, and might be separated almost completely from it, its inner layers being of a somewhat more brittle character.

Chemical Examination.—The crystalline mass of the nucleus was almost completely dissolved in ether; the reddish-brown shell likewise yielded about two-thirds of its weight to the same medium. The pale-yellow ethereal solution contained chiefly cholesterine, mixed with a very small quantity of fat, partly saponified, and partly unsaponified.

A very inconsiderable quantity of salts of the biliary acids, mixed for the most part with some yellow or green pigment, was extracted from the residue, partly by spirit of wine and partly by water.

The reddish-brown powder that remained after this treatment, yielded to chloroform a small quantity of cholepyrrhin, which gave a faint-yellowish tint to the solution. By the simultaneous addition of acids (hydrochloric acid), a large portion of the powder was dissolved; the reddish-brown color disappeared, and a dirty yellowish-brown residue remained.

The substances contained in the solution were the following:—

Cholepyrrhin in the largest proportion.

Green pigment.

Biliary acids, in distinguishable, but very small quantity.

Solid fatty acids, melting at 60° C. (140° Fahr.), in small quantity.

The subjoined inorganic bases and salts were found united with the above-mentioned organic matters:—

Lime, in by far the largest proportion, united for the most part with cholepyrrhin, and in much smaller quantity with phosphoric acid.

Magnesia in small quantity, along with the lime.

Iron, in considerable quantity.

Copper, in inferior quantity to the iron.

Traces of manganese.

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[1] The analyses were conducted, for the most part, by Dr. Neukomm.

[2] The figures referred to in the Appendix correspond to those in Frontispiece, Vol. III.—Transl.

The brownish-yellow residue that remained after repeated extraction with chloroform, and which now yielded only a small quantity of pigment to this medium, was dissolved for the most part in a spirituous solution of soda. The intense yellowish-brown solution contained brown, mixed with green, pigment.

After removal of these pigments, a reddish-yellow cholepyrrhin remained, which was only with difficulty dissolved in chloroform, and which was consequently regarded as a sparingly soluble modification of this pigment.

No. 2. Frontispiece, Vol. III., Fig. 7.

Physical Characters.—An oval concretion measuring about 12 millimètres (about 5½ Eng. lines) in its shortest, and 23 millimètres (10·8 Eng. lines) in its longest diameter. The surface was slighty rough, and was formed by a brown crust, resembling bone, one millimètre (.47 Eng. line) thick, which could be easily detached.

On its broken surface, the calculus presented a shell, from 1 to 4 millimètres (.47 to 1.89 Eng. line) thick, consisting of a soft, grayish-white mass, with a distinctly crystalline fracture, and a nucleus composed of laminæ, glistening like glass, of radiated structure and in close contact with one another; between these layers, more particularly in the centre and on the outer surface of the nucleus, a yellowish-brown substance was deposited.

The nucleus and shell passed into one another, and could with difficulty be separated.

Chemical Examination.—In the stony-hard plate deposited on the outer surface, no crystalline or bony structure of any sort could be discovered. In diluted hydrochloric acid this crust was dissolved with the evolution of a large quantity of carbonic acid, a little mucus remaining behind. The hydrochloric acid solution contained much lime, and some phosphoric acid, with traces of magnesia and iron.

The shell of the concretion yielded to ether a large quantity of colorless cholesterine.

The residue, which consisted of brownish-yellow flakes and a colored granular material, was dissolved for the most part in dilute hydrochloric acid and chloroform, with the evolution of carbonic acid. The hydrochloric acid solution contained lime, some phosphoric acid, and traces of magnesia and iron. The chloroform took up a small quantity of cholepyrrhin.

The glistening laminæ of the nucleus were entirely dissolved in ether, leaving behind only the intermediate yellowish-brown granules and crumb-like particles. The solution contained almost pure cholesterine; cholepyrrhin and lime were discovered in the residue.

No. 3. Frontispiece, Vol. III., Fig. 12.

Physical Characters.—A four-sided concretion, with rounded angles and smooth surface.

Commencing from without, there were observed, in the first place, several thinly laminated layers, of various colors, whitish yellowish, or greenish, on the whole of firm consistence, and not exceeding one millimètre (.47 Eng. line) in thickness. Within this, the layers were denser, indis-

tinctly defined, and of a greenish color. These two portions together formed an external crust from 1 to 1.5 millimètre thick (.47 to .7 Eng. line), on the inner surface of which soft and whiter deposits were again perceived.

Beneath the crust lay concentric layers of crystals disposed in a radiated manner, with yellowish-brown pigment deposited between the layers.

A cleft nucleus, with a white film of cholesterine, occupied the centre.

Chemical Examination.—The layers composing the shell, when triturated and digested with ether, yielded a small quantity of cholesterine to this medium. The residue was dissolved in dilute hydrochloric acid, with the evolution of much carbonic acid, leaving behind green flakes, which consisted, for the most part, of bile-pigment.

The hydrochloric acid solution contained a large quantity of lime, some phosphoric acid, oxide of manganese, and iron.

The loose mass forming the nucleus consisted principally of cholesterine. The amorphous masses of pigment scattered through it yielded some cholepyrrhin, in combination with lime, together with a non-nitrogenous brown substance soluble in alkalies.

No. 4. Frontispiece, Vol. III., Fig. 17.

Physical Characters.—Twelve small concretions, from the size of a millet-seed to that of a pea, their surfaces being grayish-white and smooth, but marked by slightly bulging prominences, so that some of them appeared to have almost a mulberry form. Many of the bulgings presented a depression or even an opening, through which a yellow material came to light.

On section of each of these concretions, a yellow nucleus was found, consisting of a loose mass, in which a distinct crystalline structure could be recognized. This nucleus was surrounded by a grayish-white shell, which was firm, though easily divided, and was here and there broken through.

Chemical Examination.—The shell and nucleus were dissolved almost entirely in ether; the solution was pale-yellow and was composed almost exclusively of cholesterine.

The scanty residue, consisting of yellowish-brown flakes and granules, contained a little free cholepyrrhin, and a still larger quantity in combination with lime, traces of other constituents of bile, and likewise distinguishable quantities of phosphoric acid and iron, together with some lime.

No. 5. Frontispiece, Vol. III., Fig. 6.

A large number of small rounded or oval calculi, varying in size from poppy-seeds to millet-seeds, with smooth surfaces of a satiny lustre, and mixed with a few larger bluish-white concretions presenting nodulated, but polished shining surfaces.

The smaller concretions were somewhat harder and more brittle than wax; their substance was almost uniformly firm; the larger ones only were somewhat loose and yellowish in the centre.

After digestion for several days in ether, the concretions were dissolved entirely, leaving a scarcely visible residue. The ethereal solution contained almost pure cholesterine.

The larger concretions presented on section a firm, dense, grayish-white shell, without any distinct crystalline structure, and in the interior, a yellowish-brown nucleus, composed of a loose, crystalline, laminated mass.

When digested with ether, the shell was entirely dissolved, and only a few yellowish-brown flakes of the nucleus remained. The ethereal solution, which was scarcely colored, restored the cholesterine when evaporated.

No. 6.— Gall-stones containing Mercury.

Physical Characters.—Small concretions, somewhat larger than a pea, and mulberry-shaped. Their outer surface was of a dirty grass green color, and here and there some yellowish-gray amorphous substance was deposited between the individual prominences. On crushing them, a thin, tolerably hard and brittle shell was discovered, which enclosed a loose, dark yellowish-green mass, presenting, here and there, small white metallic granules. These granules easily dropped out, and then soon coalesced into larger globules. On closer examination, they were ascertained to be metallic mercury.

Chemical Examination.—The concretions when triturated, yielded to ether a small quantity of a yellowish, greasy fat, in which no cholesterine could be discovered.

Spirit of wine dissolved a small quantity of a yellowish compound of the biliary acids with an alkali.

The residue, when treated with hydrochloric acid, yielded carbonic acid, and when chloroform was added to it immediately afterwards, it dissolved out some cholepyrrhin, while the subsequent addition of spirit of wine extracted an amorphous green pigment. The hydrochloric acid solution contained chiefly lime, small quantities of earthy phosphates, and traces of iron.

The dark-brown residue of the concretion, remaining after the above treatment, was almost completely dissolved in a solution of soda. The brown solution was precipitated by acids in the form of flakes. The scanty insoluble portion did not present the reaction of either the biliary acids or bile-pigment.

No. 7.

Physical Characters.—Concretions from the size of a pea to that of a hazel-nut, bounded partly by curved, and partly by flat surfaces, with rounded angles and edges. Isolated, warty-looking excrescences are deposited upon some of the concretions. Their surfaces were smooth, and throughout presented the appearance of iron-rust.

On section, they exhibited a dark, but by no means sharply-defined nucleus, consisting of an amorphous matter of a reddish-brown or dark-brown color. Around this nucleus were layers of a paler hue, sometimes presenting a radiated laminated structure, and at other times arranged more in concentric strata.

The darker nuclear mass was very small in some of the concretions, and formed a reddish-brown powder, which, under the microscope, presented brownish-yellow granules, together with irregularly laminated, yellow, transparent fragments. On the addition of dilute sulphuric acid, fine needles and clusters of needles of sulphate of lime immediately sep-

arated, these crystals being deposited directly upon the brownish-yellow granules and scales, which were themselves but little changed.

The paler layers deposited around the nucleus, presented, under the microscope, partly transparent laminæ, lying upon one another in large numbers and of an irregular form, and partly an amorphous yellow material interspersed in the form of granules or flakes between the laminæ.

Chemical Examination.—The greater part of the concretion was dissolved in ether. On evaporation, the pale-yellow solution left behind a considerable quantity of tolerably colorless cholesterine, to which a greasy-resinous substance still adhered in small quantity.

The yellowish-brown residue remaining after extraction by ether, yielded to chloroform small quantities of cholepyrrhin (which, upon evaporation of the solution, separated in the form of garnet-red crystals), together with some green pigment.

With the simultaneous addition of hydrochloric acid, chloroform dissolved a considerable quantity of cholepyrrhin, mixed with green pigment and traces of the biliary and fatty acids. The hydrochloric acid solution contained chiefly lime, with traces of phosphoric acid, magnesia, and iron.

The residue of the concretion remaining after the above treatment yielded to spirit of wine, some green pigment, and on the subsequent addition of a spirituous solution of soda, a yellow solution was formed, which, when treated with hydrochloric acid, likewise became green, and after evaporation deposited flakes of green pigment.

Lastly, after long boiling in moderately diluted caustic soda, the remaining portion of the concretion was almost completely dissolved, and the brown solution was precipitated by acids in the form of flakes. The insoluble portion was nitrogenous, and, when burnt, left behind but a very small quantity of ash.

No. 8.

Physical Characters.—Small dark-green concretions, somewhat larger than a pea, with uneven, rough, and occasionally indented surfaces, which at some places presented a bituminous lustre. On the application of slight pressure, the concretion broke down into irregularly-shaped fragments, which were all very brittle and black; a few pale, grayish-yellow, laminated deposits were observed here and there.

Chemical Examination.—Ether, spirit of wine and water dissolved only very sparing quantities of the triturated concretion. The ethereal solution contained isolated plates of cholesterine, together with some pigment and a greasy fat. Traces of an alkali combined with biliary acids were found in the spirituous solution.

The subsequent addition of chloroform extracted some cholepyrrhin, which upon evaporation separated in the crystalline form and was partly oxydized into cholechlorin.

After the action of hydrochloric acid, chloroform dissolved a considerable quantity of cholepyrrhin, with an admixture of only a small quantity of other substances. When spirit of wine was now added, it extracted a considerable quantity of green pigment.

The hydrochloric acid solution contained lime, some magnesia, small quantities of earthy phosphates, iron and copper.

The residue of the concretion after the above treatment, still yielded some rather insoluble cholepyrrhin to a spirituous solution of soda. Lastly,

a dark-brown residue, resembling humine, was left; this was dissolved for the most part in dilute caustic soda, leaving only a few brown flakes, which ran together on paper like little drops of resin.

On the addition of acids to the brown soda solution, colored flakes were precipitated, which could not be referred with certainty to any of the known elements of bile, but which consisted, for the most part, of organic matter containing but little nitrogen.

No. 9.

Physical Characters.—An almost cylindrical concretion, 3 centimètres (1·18 Eng. inch) in length, and 0·7 cent. (3⅛ Eng. lines) in breadth, from the biliary passages of a man, 54 years of age. This concretion was of a dark-green color, brittle and crumbling; its rough outer surface presented a powder-like film; the fresh broken surface had a bituminous lustre.

Chemical Examination.—Ether dissolved out a yellowish, oily-looking substance, which, on exposure to the air, gradually became covered with resin, presented the reaction of biliary acids in a marked degree, but contained no cholesterine.

Spirit of wine extracted from the concretion a considerable quantity of biliary acid salts. These salts were partly alkaline and partly earthy, and among the latter the compound with lime preponderated. The latter substance separated from the spirituous solution in the form of microscopic, glistening, globules with a dark contour, which bore a great resemblance to globules of leucine.

After the concretion had been extracted to exhaustion by spirit of wine, it was treated with dilute hydrochloric acid. The solution was of a faint green color and contained a tolerably large quantity of lime, some magnesia, small quantities of earthy phosphates, iron and copper.

The residue yielded to spirit of wine, besides some bile-pigment, a considerable quantity of a biliary acid which crystallized from the colorless solution in the form of bundles of delicate, silky, needles, that withered on drying. These crystals yielded the reaction of biliary acid in a marked degree. In their characters and their behaviour with reagents, they resembled glycocholic acid; but the quantity obtained was not sufficient for analysis.

The concretion, after being treated in the manner just mentioned, left behind a small quantity of a black substance, which in the moist state was soft and flaky, and in the dry resembled humine. This consisted for the most part of a nitrogenous organic matter, with a small quantity of ash.

No. 10. *Gall-stone from an Ox.*

Physical Characters.—An elongated-oval, somewhat flattened mass of from three to four inches in its long, and two inches in its broad, diameter, with an uneven, rough, and furrowed surface, which was for the most part of a dirty-brown color. Its broken surface presented a series of concentric layers of a vitreous brown substance resembling colophony, with a yellowish-brown pulverulent substance deposited here and there between the layers. Nearer the centre of the concretion, layers of a whitish, pulverulent substance were likewise observed. On microscopic examination of the last-mentioned substance, glistening flaky masses were discov-

ered, mixed with yellow pigment, either in the form of granules or generally diffused.

Chemical Examination.—The ethereal extract of a portion of the triturated concretion contained a crystalline unsaponified fat, with free fatty acids and free biliary acids. No cholesterine could be discovered in it.

Spirit of wine and water dissolved out a considerable quantity of alkaline salts of the biliary acids, mixed with chloride of sodium, pigment, and free biliary acid. The last-mentioned substance separated from the concentrated spirituous solution, on the addition of water; it was partly amorphous, resembling resin, and partly crystalline. The purified crystals proved to be cholic acid.

The following substances were found united with inorganic bases, and were only extracted by means of chloroform and spirit of wine, after treatment with hydrochloric acid:—

Cholepyrrhin in considerable quantity.

Green pigment.

Stearic acid in small quantity.

The hydrochloric acid solution contained lime, some magnesia, and a small quantity of earthy phosphates.

The residue of the concretion, which was still considerable, consisted almost exclusively of the particles of the triturated vitreous layers. These particles became swollen up in water, without dissolving; they were homogeneous throughout, and devoid of any organic or crystalline structure; in other particulars they conducted themselves like protein principles.

No. 11. *Bilio-Intestinal Concretion from an Ox.*

Physical Characters.—A nodulated yellowish-white body, bearing a great resemblance to rhubarb-root. The broken surface presented alternating layers of vegetable substances (such as straw, husks, &c.), and of a firm yellow or white granulo-pulverulent material, interspersed here and there with layers of a more brownish color.

Under the microscope, the white chalky pulverulent layers presented needles, either arranged in a confused manner or grouped together in bundles, and broken off for the most part at one extremity, whilst in other parts of the concretion, and more particularly in the yellower layers, glistening flaky masses, with a few needles, were discovered. A brownish-yellow pigment was partly diffused through isolated flakes and partly interspersed in the form of granules and irregular particles, but its amount at most places was scanty.

Chemical Examination.—The following unequivocal elements of bile were obtained from the concretions by chemical reagents:—

Cholic acid was extracted by ether, but mainly by spirit of wine, in considerable quantity.

Resinous biliary acids, in small quantity, along with the cholic acid.

Calcareous compounds of cholic acid. They were dissolved in largest quantity in spirit of wine, far more sparingly in water. From the spirituous solution they crystallized in the form of elongated, pointed needles, partly arranged in a confused manner, and partly united in bundles. White crusts separated on the surface from the watery solution. When dried, this substance formed a white, easily triturated, light substance.

The lime-salt of another biliary acid, which was distinguished from cholate of lime, by being precipitated in the form of microscopic globules, with a dark contour, and not unfrequently of mulberry-formed surface. Both compounds of lime occurred mixed with one another.

Bile-pigments were found mixed in small quantity with the substances contained in the ethereal and spirituous extracts, but they were not examined more minutely.

Solid fatty acids were found in the ethereal extract along with cholic acid; they crystallized from spirit of wine in the form of laminated crystalline masses.

Cholesterine was searched for without any result.

The residue of the concretion, after removal of the above-mentioned elements, was inconsiderable. It contained an ash, composed of carbonate of lime, some magnesia, small quantities of earthy phosphates, a rather large amount of iron and alumina, and likewise silicic acid, which was partly mixed with the other constituents of the calculus in the form of sand, and partly contained in the vegetable ingredients.

No. 12.

A concretion from the Pathological Collection at Göttingen, composed mainly of margarate of lime, was examined by me in 1847.

It was of an oval form, smooth and brownish-yellow. The dazzling white broken surface had a striated crystalline structure, and in the centre was a brown, cleft nucleus, the size of a lentil. The substance of the concretion was easily triturated, but was only sparingly dissolved in boiling ether and spirit of wine. The spirituous solution, on evaporation, deposited plates of cholesterine, small globules and needle-shaped crystals. The portion of the concretion, which was insoluble in spirit of wine, yielded to dilute acetic acid a large quantity of lime, and then dissolved readily in boiling spirit of wine. On evaporation, the spirit deposited minute crystalline scales, which, when purified, melted at a temperature of 58° C. (136°.4 Fahr.). The nucleus consisted of the compound of cholepyrrhin and lime, and of mucus.

The concretion had the following composition in 100 parts:—

Cholesterine	28·04
Margarate of Lime	68·56
Mucus and the Compound of Cholepyrrhin and Lime	3·40
	100·00

II.—Experiments on the Excretion of Hippuric Acid in Jaundice. By Dr. Neukomm.

From the experiments made by Kühne and Hallwachs upon dogs and cats, these observers drew the conclusion that the formation of hippuric acid in the blood is entirely dependent upon the presence in it of the elements of bile, and that therefore, under normal circumstances, it only takes place within the hepatic circulation.[1] An observation that Kühne subsequently believed he had made in a person affected with jaundice, according to which not a trace of hippuric acid was found in 2 litres (70·43

[1] *Arch. f. Path. Anat.*, Bd. XII.

fluid ounces) of the urine of a patient, who had taken from 6 to 8 grammes (3 jss. to 3 ij.) of benzoic acid, led him to the conclusion that in jaundice consequent on closure of the ductus choledochus, no hippuric acid whatever is formed.[1]

Kühne, however, in the very same patient, and at the same time that he maintained that not a trace of hippuric acid was found after taking benzoic acid, discovered a constant and not inconsiderable quantity of biliary acids in the urine, which of course necessitated the pre-existence of this substance in the blood. As this observation was completely opposed to the statement formerly made by himself and Hallwachs, the perfectly gratuitous assumption was framed, that certain elements of the bile, such as tauro-cholic acid, &c., are constantly formed in the liver, but that the production of glyco-cholic acid or glycin is completely suspended in jaundice.[2]

Although this is not the place to discuss the qualitative and quantitative composition of human bile, still less to consider the manner in which this secretion becomes altered by closure of the ductus choledochus, the alteration of the urine observed by Kühne, appeared to us sufficiently important to be made the subject of further experiment.

Accordingly, ten grains of benzoic acid, in two doses, were administered in the evening to a young man, who had suffered from jaundice for several weeks, and whose fæces for the last few days had been totally devoid of color. The urine passed during the night and on the following morning, which differed in no essential particular from that previously secreted (being of an intense yellowish-brown color, of a feeble alkaline reaction, and yielding with nitric acid the reaction of bile-pigment in a marked degree), was tested for hippuric acid.

For this purpose, it was evaporated and extracted with spirit of wine. The spirituous solution was reduced to a syrup, which was strongly acidified with hydrochloric acid and then agitated in a tube with a quantity of ether. The ethereal solution contained the largest quantity of hippuric acid that could be expected to be present; it was evaporated, and the hippuric acid was dissolved out of the resinous residue by water. On evaporation of the water, the acid separated in numerous characteristic crystals, which were proved to be hippuric acid, by their chemical reaction.

Some days after, when the patient was still passing stools perfectly devoid of color, the experiment was repeated in like manner. The result exactly corresponded with that obtained in the first instance.

In both experiments, the quantity of hippuric acid obtained did not exceed one grain.

After the patient had been for several days without taking any more benzoic acid, the urine was again tested for hippuric acid. The quantity obtained was perceptibly less then after taking the benzoic acid, but did not differ essentially from the amount found at the same time in an equal volume of non-jaundiced urine.

The patient died subsequently; death being preceded by cerebral symp-

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[1] *Ibid.*, Bd. XIV.

[2] Kühne employs this argument to refute Frerichs's theory of jaundice. Frerichs maintains that the pigment in jaundice is derived from a transformation of the colorless biliary acids absorbed into the blood. (See Vol. I., Translator's Preface, p. xii.) Kühne asserts, on the other hand, that in jaundice the formation of the biliary acids, or of the most important of them, is suspended, and that this is proved by the non-conversion of benzoic acid into hippuric acid.—TRANSL.

toms. The urine passed during the last comatose stage, when the amount of nourishment taken was almost *nil*, likewise contained a considerable quantity of hippuric acid.

In another individual affected with jaundice, who had never taken any benzoic acid, the quantity of hippuric acid found in the urine did not differ remarkably from that contained in non-jaundiced urine collected under like circumstances.

III.—List of Observations on Diseases of the Liver.

No. XVIII. (*Page* 60.) Dipsomania and irregular habits of life—Persistent derangements of digestion—Jaundice—Enlarged liver—Somnolence—Noisy delirium—Coma—Death.

Autopsy: Enlarged liver, with circumscribed masses of inflamed tissue scattered through it—Destruction of the secreting cells, and hypertrophy of the areolar framework—Small spleen—Extravasations of blood in the lungs, and beneath the pleura and the epicardium—Fatty degeneration of the muscular tissue of the heart and of the kidneys—Urine abounding in Tyrosine, Kreatine, and Leucine, and emitting an odor of sulphuretted hydrogen.

No. XIX. (*Page* 64.) Pains in the epigastrium—Vomiting—Slight fever—Enlarged liver—No tumefaction of spleen—Jaundice—Petechiæ—Hæmatemesis—Somnolence—Death.

Autopsy: Large, fatty, and jaundiced liver, with disintegrating cells and pervious bile-ducts—Ecchymoses beneath the pleura and epicardium—Small spleen—Fatty kidneys.

No. XX. (*Page* 65.) Symptoms of acute gastric catarrh, with great fever—Somnolence—Coma—Noisy delirium—No tumefaction of the spleen—Jaundice—Urine abounding in Tyrosine and Kreatine—Death from cerebral paralysis.

Autopsy: Softening of the liver—Disintegration of the glandular cells, and commencing atrophy—Kidneys soft, and in a state of fatty degeneration—Spleen of normal size, and congested.

No. XXI. (*Page* 67.) Fifth month of pregnancy—Bilious vomiting—Constipation—Violent headache, increasing so as to cause loss of consciousness—Enlarged and painful liver—Tumefaction of the spleen—Albuminuria—Slight Jaundice—Cure.

No. XXIII.[1] (*Page* 92.) Extensive ascites without œdema of the feet—Disordered gastric and intestinal digestion—Urgent dyspnœa—No obvious cause for the disease—Temporary improvement—Increase of the dropsy—Administration of purgatives—Paracentesis—Death.

Autopsy: Cirrhosis of the liver—Thickening of the walls of the vena portæ—Splenic tumor—Fatty degeneration of the muscular tissue of the heart—Sugar and Leucine in the ascitic fluid.

No. XXIV. (*Page* 93.) Disordered gastric digestion—Vomiting—Diarrhœa—Ascites—Œdema of the feet—Puncture of the abdomen—Splenic tumor—Liver small, with nodulated surface—Death.

Autopsy: Cirrhotic and lobulated liver—Thickening of Glisson's capsule—Firm adhesion of the lower surface of the liver to the ad-

[1] There is no Observation XXII. The numbers are retained as in the original, for the convenience of reference.—Transl.

joining parts, and also of the indurated pancreas to the vertebral column and retro-peritoneal glands—Recent peritonitis.

No. XXV. (*Page* 95.) Paralysis (*Lähmung*) of the hypoglossal and facial nerves, and incomplete paralysis (*Parese*) of the muscles of the trunk and extremities—Dysentery—General convulsions—Death.

Autopsy: Enlargement and abnormal mobility of odontoid process of second vertebra—Granular induration of liver without any obvious cause—Splenic tumor—Slight ascites—Dysenteric inflammation of the large intestine.

No. XXVI. (*Page* 97.) Persistent intermittent fever—Irregular habits of life—Gastric catarrh—Slight jaundice—Cachexia—Ascites—Paracentesis—Collapse—Death.

Autopsy: Finely granular cirrhosis of liver—Splenic tumor, with slight pigment-deposit—Catarrhal tumefaction of the mucous membrane of the stomach—Cicatrices in the duodenum—Typhus (*sic*) cicatrices in the ileum.

No. XXVII. (*Page* 100.) Intermittent fever of seven months' duration—Ascites—Hydræmia—Anasarca—Death from œdema of the lungs.

Autopsy: A moderately enlarged pigment-spleen—Cirrhosis of the liver—Mucous membrane of the stomach and intestines, and the kidneys normal.

No. XXVIII. (*Page* 101.) Old pleuritic exudation—Persistent intermittent fever—Tubercle of both lungs—Ascites—Bronzed skin—Small liver—Symptoms of indigestion.

Autopsy: Firm adhesions of pleuræ—Tubercle of the lungs—Cirrhosis of the liver—Supra-renal capsules normal.

No. XXIX. (*Page* 102.) Previous syphilis—Abuse of spirits—Double pneumonia—Death from œdema of the lungs.

Autopsy: Inflammatory exudation in both lungs—Cirrhosis of the liver—Moderate tumefaction of the spleen—No ascites, and no gastro-enteric catarrh.

No. XXX. (*Page* 103.) Constitutional syphilis—Repeated courses of mercury—Albuminuria—Splenic enlargement—Right pleurisy—Dropsy—Death from acute enteric catarrh.

Autopsy: Amyloid degeneration of the kidneys, spleen, and liver—Cirrhotic shrivelling and lobulation (*Lappung*) of the liver—Purulent effusion in the right pleura—Cicatrices and old ecchymoses of the stomach—Catarrhal inflammation of the small intestine.

No. XXXI. (*Page* 105.) Constitutional syphilis—Systolic bruit over apex of heart—Dyspnœa—Cyanosis—Large spleen—Bulging, nodulated liver—Albuminuria—General dropsy.

Autopsy: Incompetence of the mitral valves—Lardaceous degeneration of the liver, spleen, and kidneys—Cicatrices and cirrhotic degeneration of the liver—Obliteration of a portion of the portal vessels—Remarkable increase of the white blood-corpuscles in the portal and hepatic veins.

No. XXXII. (*Page* 107.) Constitutional syphilis—Hæmoptysis—Dulness and consonant râles over the apex of the left lung—Ascites—Albuminuria—Tenderness and slight dulness in the region of the liver—Thin, pale stools.

Autopsy: Small, cirrhotic, indurated liver—Moderately large lardaceous spleen—Syphilitic disease of the cranial bones—Tubercle at the apices of both lungs—Granular kidneys.

No. XXXIII. (*Page* 108.) Constitutional syphilis—Epilepsy—Abuse of spirits—Death in an epileptic fit.

Autopsy: Cirrhosis of the liver—Enlarged spleen—Chronic catarrh of the stomach.

No. XXXIV. (*Page* 109.) Jaundice of 18 months' duration—Enlarged liver, with uneven surface—Death under symptoms of acute peritonitis.

Autopsy: Granular lardaceous liver—Lardaceous spleen—Infiltration of the glands in the fossa hepatis, and in the inguinal region—Purulent peritoneal exudation.

No. XXXV. (*Page* 111.) Abdomen enlarged and painful—Deranged Digestion—Ascites—Enlargement of the spleen—Surface of the liver felt covered with nodules—Paracentesis—Profuse watery Diarrhœa—Exhaustion—Death.

Autopsy: Lobulated cirrhotic liver—Enlarged spleen—Mucous membrane of the stomach and intestines livid and much relaxed.

No. XXXVI. (*Page* 114.) Abuse of spirits—Apoplectic attack—Temporary painful enlargements of the liver during six years—Jaundice—Dyspnœa—Bloody stools with tenesmus—Slight somnolence—Much albumen and kreatine, and traces of the biliary acids, in the urine.

Autopsy: Cirrhotic induration of the liver—Hepatic cells partly destroyed—Dysenteric disease in the small and large intestine—Pneumonia—Cysticerci in the brain and in the thoracic muscles.

No. XXXVII. (*Page* 122.) Abdominal pain—Circumscribed peritoneal exudation—Slight jaundice—Improvement—Six months afterwards, extensive ascites—Œdema of the lower half of the body—Gangrenous erysipelas—Death.

Autopsy: Remains of old and recent peritonitis—Thickening of the mesentery—Numerous adhesions of the spleen and liver—Hæmorrhage from the stomach and intestines—A moderately enlarged spleen—Granular and simple induration of the liver—Constriction of the hepatic veins.

No. XXXVII. (*bis*).[1] (*Page* 164.) Chronic bronchial catarrh—Ozæna syphilitica—Cicatrices on the velum palati—Liver covered with deep fissures and nodulated projections, and, at some places, painful.

No. XXXVIII. (*Page* 165.) Deranged digestion—Cachectic appearance and debility—Anasarca without albuminuria—Catarrh—Liver enlarged, deformed, and tender upon pressure—Splenic tumefaction—Death from œdema of the lungs.

Autopsy: Cicatrices in the pharynx and œsophagus—Catarrh of the air-tubes—Remains of peri-hepatitis and hepatitis gummosa, together with circumscribed amyloid infiltration—Firm splenic tumor—Kidneys normal.

No. XXXIX. (*Page* 167.) Persistent vomiting of mucous matter—Œdema of the feet—Albuminuria—Syphilitic cicatrices upon the forehead—Indurated chancre upon the genital organs—Bloody stools—Death.

Autopsy: Lobulation and induration of the liver from syphilitic cicatrices—Obliteration of numerous branches of the portal vein—Amyloid degeneration of the spleen (which was small), and of

[1] There are two Observations marked No. XX. (now No. XXXVII.) in the original—TRANSL.

the kidneys—Hæmorrhage from the mucous membrane of the small and large intestines.

No. XL. (*Page* 168.) Syphilis many years before—At a later period, symptoms of pulmonary consumption—Albuminuria—Diarrhœa—Dropsy—Death from exhaustion.

Autopsy: Syphilitic caries of the cranial bones—Thickening of the dura mater—Cicatrices in the pharynx—Tubercles at the apices of both lungs—Deformed waxy liver with syphilitic cicatrices—Waxy spleen and waxy kidneys—Amyloid matter in the mucous membrane of the small intestine.

XLI. (*Page* 183.) Necrosis of the femur—Repeated syphilitic infection—Secondary symptoms—Several courses of mercurial treatment—Albuminuria—Enlargement of the spleen and liver—Anasarca—Improvement under iodide of iron—Relapse—Aggravation of symptoms in consequence of inappropriate treatment—Renewed use of iodide of iron—Chalybeates and warm baths—Cure.

No. XLII. (*Page* 184.) Secondary syphilis—Abuse of mercury—Pseudo-rheumatic pains—Jaundice—Tumefaction of the liver and spleen—Cure by drinking and bathing in the mineral waters at Aix-la-Chapelle, with the use of iodide of potassium internally.

No. XLIII. (*Page* 185.) Syphilitic disease of the bones—Syphilitic ulcers of the mucous membrane of the nostrils—Pains in the larynx—Impending asphyxia—Tracheotomy—Death.

Autopsy: Stricture of the larynx—Lardaceous liver—Enlarged spleen—Fatty kidneys.

No. XLIV. (*Page* 187.) Hæmatemesis—Distention and tenderness of the hepatic region—Jaundice—Thin pale stools—Feeble action of the heart—Dyspnœa—Sudden death under symptoms of asphyxia.

Autopsy: Infarctions of the lungs—Thrombi in the pulmonary artery—Simple ulcer and cicatrices in the stomach—Waxy and fatty degeneration of the liver—Small spleen—Normal kidneys—Ulceration and osteophytes of the skull-cap—cicatrices in the vagina.

No. XLV. (*Page* 188.) Syphilitic infection years before—Epithelial cancer of the penis—Amputation of penis—Albuminuria—Dropsy—Right pleurisy and œdema of the lungs of a threatening character—Bloody urine—Diarrhœa—Urine at first abundant, and afterwards scanty—Gangrenous erysipelas—Death.

Autopsy: Amyloid degeneration of the kidneys, spleen, and liver—Purulent effusion into the cavity of the pleura—Cicatrices in the pharynx—Old thrombus in the left renal vein.

No. XLVI. (*Page* 190.) Carious ulceration of the hip-joint and necrosis of the femur of many years' duration—Enlargement of the liver and spleen—Albuminuria—General dropsy—Protracted use of cod-liver oil in large doses.

Autopsy: Large waxy liver, with deposit of fat—Waxy spleen (sago-spleen, *Sagomilz*), and waxy kidneys.

No. XLVII. (*Page* 191.) Rickets—Tumefaction of the spleen and liver—Death from bronchitis and lobular pneumonia.

Autopsy: Rachitic disease of the cranial bones, the ribs, and the bones of the legs—Lobular pneumonia—Waxy spleen—Fatty liver with waxy degeneration—Enlargement of the mesenteric glands.

No. XLVIII. (*Page* 191.) Persistent intermittent fever—Uniform firm

enlargement of the liver and spleen—Dissipated habits—Pneumonia of right Lung—Intoxication from liquor ammoniaci caustici—Pharyngitis—Pneumonia of left Lung—Death.

Autopsy: Waxy degeneration of the liver and spleen—Inflammatory infiltration of both lungs.

No. XLIX. (*Page* 193.) Persistent intermittent fever—Typhus—Tumefaction of the upper region of the abdomen—Vomiting—Diarrhœa—Œdema of the feet—Large smooth liver, and enlarged spleen.

Autopsy: Waxy degeneration of the liver—Great tumefaction of the spleen—Pneumonia ultima.

No. L. (*Page* 196.) Chronic tubercle of the lungs and intestines—Treatment by cod-liver-oil—Waxy and fatty degeneration of the liver—Commencing degeneration of the spleen—Kidneys normal.

No. LI. (*Page* 197.) Symptoms of pulmonary and laryngeal phthisis—Diarrhœa—Large, firm tumefaction of the liver—Ascites.

Autopsy: Tubercular deposits in the larynx, trachea, lungs and intestines—Very large fatty liver, with circumscribed waxy degeneration — Soft spleen, with isolated waxy deposits—Fatty kidneys.

No. LII. (*Page* 198.) Hæmorrhages from the vagina and stomach—Cancer of the uterus and of the cardiac orifice of the stomach—Tumefaction of the spleen and liver—Death from exhaustion.

Autopsy: Cancer of the uterus and of the cardia—Amyloid degeneration of the liver and spleen—Dilatation of the calices of the kidneys—Renal calculi.